

=> fil reg
COST IN U.S. DOLLARS

FULL ESTIMATED COST

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

CA SUBSCRIBER PRICE

SINCE FILE
ENTRY
1.78

TOTAL
SESSION
1096.16

SINCE FILE
ENTRY
0.00

TOTAL
SESSION
-76.72

Apwack
486823
pt 1 of 3

FILE 'REGISTRY' ENTERED AT 14:31:10 ON 04 SEP 2001
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2001 American Chemical Society (ACS)

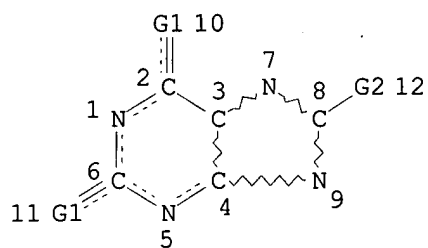
STRUCTURE FILE UPDATES: 3 SEP 2001 HIGHEST RN 354528-22-6
DICTIONARY FILE UPDATES: 3 SEP 2001 HIGHEST RN 354528-22-6

TSCA INFORMATION NOW CURRENT THROUGH January 11, 2001

Please note that search-term pricing does apply when
conducting SmartSELECT searches.

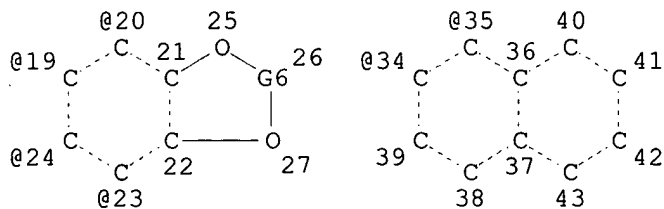
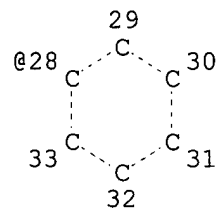
Structure search limits have been increased. See HELP SLIMIT
for details.

=> d l4 que stat;fil caplus;e neurodegenerative disease/ct 5
L1 STR



CH2G3-Hy
@13 14 15

C=C-G4
@16 17 18



VAR G1=O/S
VAR G2=13/HY/16
REP G3=(0-3) CH2
VAR G4=28/35/34/20/19/24/23
REP G6=(1-3) CH2
NODE ATTRIBUTES:
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS 43

STEREO ATTRIBUTES: NONE
L4 1960 SEA FILE=REGISTRY SSS FUL L1

100.0% PROCESSED 53274 ITERATIONS
SEARCH TIME: 00.00.29

1960 ANSWERS

COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	136.66	1232.82
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE	TOTAL
	ENTRY	SESSION
CA SUBSCRIBER PRICE	0.00	-76.72

FILE 'CAPLUS' ENTERED AT 14:38:03 ON 04 SEP 2001
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2001 AMERICAN CHEMICAL SOCIETY (ACS)

Copyright of the articles to which records in this database refer is held by the publishers listed in the PUBLISHER (PB) field (available for records published or updated in Chemical Abstracts after December 26, 1996), unless otherwise indicated in the original publications.

FILE COVERS 1947 - 4 Sep 2001 VOL 135 ISS 11
FILE LAST UPDATED: 3 Sep 2001 (20010903/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

This file supports REGISTRY for direct browsing and searching of all substance data from the REGISTRY file. Enter HELP FIRST for more information.

CAPLUS now provides online access to patents and literature covered in CA from 1947 to the present. On April 22, 2001, bibliographic information and abstracts were added for over 2.2 million references published in CA from 1947 to 1966.

The CA Lexicon is now available in the Controlled Term (/CT) field. Enter HELP LEXICON for full details.

Attention, the CA Lexicon is the copyrighted intellectual property of the American Chemical Society and is provided to assist you in searching databases on STN. Any dissemination, distribution, copying, or storing of this information, without the prior written consent of CAS, is strictly prohibited.

E#	FREQUENCY	AT	TERM
---	-----	--	----
E1	0	2	NEURODEGENERATION/CT
E2	0	1	NEURODEGENERATIVE/CT
E3	0	2 -->	NEURODEGENERATIVE DISEASE/CT
E4	826	2	NEURODEGENERATIVE DISEASES/CT
E5	0	2	NEURODEGENERATIVE DISEASES (L) NIGROSTRIATAL DEGENERAT ION/CT

=> e e3+all/ct

E1	0	-->	Neurodegenerative disease/CT
E2		USE	Nervous system (L) degeneration/CT
***** END***			

=> e neurodegenerative diseases/ct 5

E#	FREQUENCY	AT	TERM
---	-----	--	----
E1	0	1	NEURODEGENERATIVE/CT
E2	0	2	NEURODEGENERATIVE DISEASE/CT
E3	826	2 -->	NEURODEGENERATIVE DISEASES/CT
E4	0	2	NEURODEGENERATIVE DISEASES (L) NIGROSTRIATAL DEGENERAT ION/CT

E5 0 2 NEURODEGENERATIVE DISEASES (L) NIGROSTRIATUM
DEGENERAT
 ION/CT

=> e e3+all/ct

E1 826 --> Neurodegenerative diseases/CT
 HN Valid heading during volumes 126-130 (1997-June 1999)
 only.
E2 NEW Nervous system (L) degeneration/CT
***** END***

=> s e1

L5 826 "NEURODEGENERATIVE DISEASES"/CT

=> fil medl,caplus,biosis,embase;s l4 and (l5 or nervous system(l)degenerat?
or neurodegenerat?(2a)(disease? or disorder?))

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	2.53	1235.35

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-76.72

FILE 'MEDLINE' ENTERED AT 14:39:39 ON 04 SEP 2001

FILE 'CAPLUS' ENTERED AT 14:39:39 ON 04 SEP 2001
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2001 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'BIOSIS' ENTERED AT 14:39:39 ON 04 SEP 2001
COPYRIGHT (C) 2001 BIOSIS(R)

FILE 'EMBASE' ENTERED AT 14:39:39 ON 04 SEP 2001
COPYRIGHT (C) 2001 Elsevier Science B.V. All rights reserved.

L6 1 FILE MEDLINE
L7 3 FILE CAPLUS
L8 3 FILE BIOSIS
L9 4 FILE EMBASE

TOTAL FOR ALL FILES

L10 11 L4 AND (L5 OR NERVOUS SYSTEM(L) DEGENERAT? OR
NEURODEGENERAT?(2A
)(DISEASE? OR DISORDER?))

=> dup rem l10

PROCESSING COMPLETED FOR L10

L11 8 DUP REM L10 (3 DUPLICATES REMOVED)

=> d cbib abs 1-8;s (alzheimer? or parkinson?) and l4

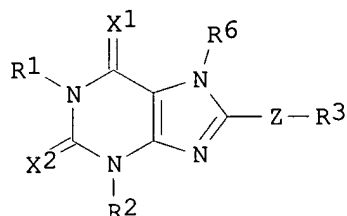
L11 ANSWER 1 OF 8 CAPLUS COPYRIGHT 2001 ACS

2001:360002 Document No. 134:366889 Preparation of polycycloalkylpurines as adenosine receptor antagonists. Kiesman, William F.; Dowling, James E.; Ensinger, Carol L.; Kumaravel, Gnanasambandam; Petter, Russell C.; Chang, He Xi; Lin, Ko Chung (Biogen, Inc., USA). PCT Int. Appl. WO 2001034610

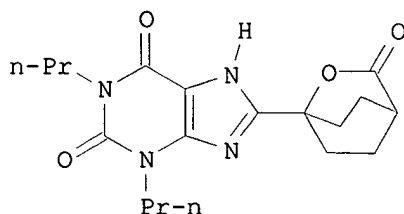
Al

20010517, 124 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2000-US31058 20001113. PRIORITY: US 1999-PV165191 19991112.

GI



I



II

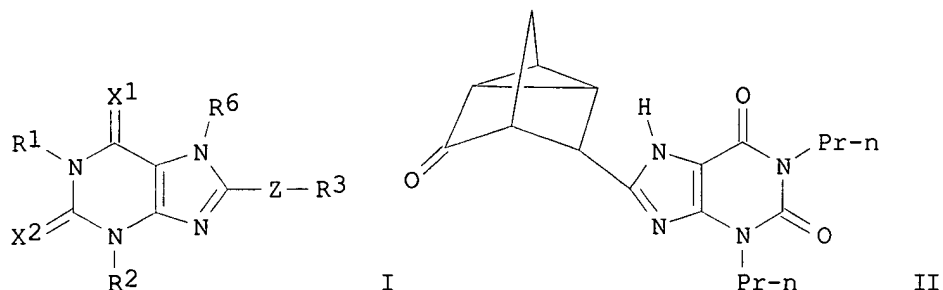
AB The title compds. [I; R1, R2 = H, alkyl, alkenyl, etc.; R3 = (un)substituted bicyclic, tricyclic, pentacyclic; X1, X2 = O, S; Z = a single bond, O, CH2OCH2, etc.; R6 = H, allyl, acyl, etc.] which are unexpectedly highly potent and selective inhibitors of the adenosine A1 receptor, and therefore can be useful in the prevention and/or treatment of numerous diseases, including cardiac and circulatory disorders, **degenerative disorders of the central nervous system**, respiratory disorders, and many diseases for which diuretic treatment is suitable, were prep'd. E.g., a multi-step synthesis of the purine II was given. All of the compds. I tested exhibited rat A1 Ki values between 0.6 and 433.8 nM, human A1 Ki values between 1.6 and 1000 nM, and human A2a Ki values between 132 and 49930 nM.

L11 ANSWER 2 OF 8 CAPLUS COPYRIGHT 2001 ACS

2001:359996 Document No. 134:366887 Preparation of 8-substituted xanthines as adenosine receptor antagonists. Dowling, James E.; Ensinger, Carol; Kumaravel, Gnanasambandam; Petter, Russell C. (Biogen, Inc., USA). PCT Int. Appl. WO 2001034604 A2 20010517, 61 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2000-US31100 20001113.

PRIORITY: US 1999-PV165283 19991112.

GI



AB The title compds. [I; R1, R2 = H, alkyl, alkenyl, etc.; R3 = (un)substituted bicyclic or tricyclic group; X1, X2 = O, S; Z = a single bond, O, (CH2)1-3, etc.; R6 = H, alkyl, acyl, etc.] which are unexpectedly highly potent and selective inhibitors of the adenosine A1 receptor, and therefore are useful in the prevention and/or treatment of numerous diseases, including cardiac and circulatory disorders, **degenerative disorders of the central nervous system**, respiratory disorders, and many diseases for which diuretic treatment is suitable, were prepd. E.g., a 2-step synthesis of II was given. All of the compds. I tested exhibited rat A1 Ki values between 0.47 and 1225 nM, human A1 Ki values between 12 and 1000 nM, and human A2a Ki values between 18 and 100,000 nM.

L11 ANSWER 3 OF 8 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
2001170591 EMBASE Neurotoxicity as a mechanism for **neurodegenerative disorders**: Basic and clinical aspects. Danysz W.. W. Danysz, Preclinical R and D, Merz Co., Eckenheimer Landstrasse 100-104, 60318 Frankfurt am Main, Germany. wojciech.danysz@merz.de. Expert Opinion on Investigational Drugs 10/5 (985-989) 2001.
Refs: 10.
ISSN: 1354-3784. CODEN: EOIDER. Pub. Country: United Kingdom. Language: English. Summary Language: English.

AB This three day meeting focused on chronic **neurodegenerative diseases** such as Parkinson's disease (PD), Alzheimer's disease (AD) and amyotrophic lateral sclerosis (ALS). It attracted 69 participants from 10 countries with dominance of Chile and USA. Neurodegeneration and its prevention increasingly gain in importance as the number of people affected increases year-by-year. The meeting addressed various basic aspects having pragmatic implications such as: oxidative stress, inflammatory reaction, glial activation, role of glutamatergic system and apoptosis using a plethora of in vitro and in vivo methods.

L11 ANSWER 4 OF 8 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 1
2001:297473 Document No.: PREV200100297473. Dual actions of A2A adenosine

receptor antagonists on motor dysfunction and neurodegenerative processes.

Ongini, Ennio; Monopoli, Angela (1); Impagnatiello, Francesco; Fredduzzi, Silva; Schwarzschild, Michael; Chen, Jiang-Fan. (1) Schering-Plough Research Inst., San Raffaele Science Park, Via Olgettina 58, 20132,

Milan:

angela.monopoli@spcorp.com Italy. Drug Development Research, (Jan Feb, 2001) Vol. 52, No. 1-2, pp. 379-386. print. ISSN: 0272-4391. Language: English. Summary Language: English.

AB Of the four known adenosine receptors, the A2A receptor has received much attention over the last few years. The discovery of high-affinity and selective A2A adenosine receptor antagonists, together with the development of different genetic lines of mice lacking A2A receptors,

have

greatly contributed to the new insights into the mechanisms whereby A2A receptors modulate central nervous system functions. Efforts made using the prototypic A2A receptor antagonists, e.g., the 8-styrylxanthine KW 6002 and the pyrazolotriazolopyrimidine SCH 58261, have shown that these drugs are effective in different models of motor impairment mimicking the main features of Parkinson's disease. Moreover, these drugs show neuroprotective properties in models of brain injury. Consistent with pharmacology, A2A receptor knockout mice have been found to be less sensitive to both motor impairment and neurochemical changes relevant to **neurodegenerative disorders**. The main effect of A2A receptor blockade or inactivation is related to selective interaction

with

dopamine-mediated function in the striatum. However, there are responses which appear to be independent of dopamine receptors while the mechanisms underlying neuroprotection remain to be elucidated. Overall, there are

now

compounds that appear to be promising for treatment of Parkinson's **disease** and related **neurodegenerative disorders**

. The efforts currently ongoing to understand their efficacy in patients will make it possible to assess whether A2A receptor blockers are a new interesting class of antiparkinsonian agents.

L11 ANSWER 5 OF 8 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.

2001155086 EMBASE Selective adenosine A(2A) receptor antagonists. Ongini E.; Monopoli A.; Cacciari B.; Giovanni Baraldi P.. A. Monopoli, Schering-Plough Res. Institute, San Raffaele Science Park, Via Olgettina 58, 20132 Milan, Italy. angela.monopoli@spcorp.com. Farmaco 56/1-2 (87 - 90) 1 Mar 2001.

Refs: 20.

ISSN: 0014-827X. CODEN: FRMCE8.

Publisher Ident.: S 0014-827X(01)01024-2. Pub. Country: France. Language: English. Summary Language: English.

AB In the early 1990s it became clear that the A(2A) adenosine receptor had characteristics that made it distinct from the other A(1), A(2B) and A(3) adenosine receptors. Great progress has been made with the discovery of selective A(2A) receptor antagonists. A variety of synthetic

substitutions

on the xanthine moiety led the chemists of Kyowa-Hakko to discover that introduction of the styryl group in the 8 position of xanthines was critical in achieving compounds endowed with selective A(2A) receptor antagonistic properties. One compound, KW 6002, (E)1,3-diethyl-8-(3,4-

dimethoxystyryl)-7-methylxanthine, is currently being developed for treatment of Parkinson's disease. A number of non-xanthine heterocycles have also been synthesized starting from the non-selective adenosine antagonist CGS 15943, a triazoloquinazoline. Thus, replacement of the phenyl ring of CGS 15943 with a heterocyclic ring such as pyrazole or imidazole, led to a series of interesting compounds whose prototype, SCH 58261, 7-(2-phenylethyl)-5-amino-2-(2-furyl)-pyrazolo[4,3-e]-1,2,4-triazolo[1,5-c]pyrimidine, has become a reference A(2A) receptor antagonist. Modification of N7 substituents has progressed to optimize A(2A) receptor selectivity and pharmacokinetic characteristics. A related class of compounds having a bicyclic instead of the tricyclic ring structure is also of interest. The prototype of these triazinotriazolo derivatives, ZM 241385, is a potent A(2A) receptor antagonist; however,

it

also shows interactions with A(2B) receptors. The relevance of the A(2A) receptors in specific disease states, especially in the central nervous system, makes this class of adenosine receptor blockers of interest for treatment of **neurodegenerative disorders** such as Parkinson's disease. Copyright .COPYRG. 2001 Elsevier Science S.A.

L11 ANSWER 6 OF 8 BIOSIS COPYRIGHT 2001 BIOSIS

2000:372814 Document No.: PREV200000372814. Anti-parkinsonian effects of selective adenosine A2A receptor antagonists in relevant rodent models. Ongini, E. (1); Impagnatiello, F. (1); Besana, C. (1); Fredduzzi, S. (1); Monopoli, A. (1). (1) Schering-Plough Research Institute, San Raffaele Science Park, Milan Italy. European Journal of Neuroscience, (2000) Vol. 12, No. Supplement 11, pp. 134. print. Meeting Info.: Meeting of the Federation of European Neuroscience Societies Brighton, UK June 24-28, 2000 ISSN: 0953-816X. Language: English. Summary Language: English.

L11 ANSWER 7 OF 8 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.

95367106 EMBASE Document No.: 1995367106. Current developments of A(2a) adenosine receptor antagonists. Baraldi P.G.; Cacciari B.; Spalluto G.; Borioni A.; Vizziano M.; Dionisotti S.; Ongini E.. Dipto. di Scienze Farmaceutiche, Universita di Ferrara, Via Fossato di Mortara 17-19, 44100 Ferrara, Italy. Current Medicinal Chemistry 2/3 (707-722) 1995. ISSN: 0929-8673. CODEN: CMCHE7. Pub. Country: Netherlands. Language: English. Summary Language: English.

AB Adenosine regulates a wide range of physiological functions through specific cell membrane receptors. On the basis of pharmacological studies and molecular cloning, four distinct adenosine receptors have been identified and classified as A1, A(2a), A(2b) and A3. These adenosine receptors are members of the G-protein-coupled receptor family. An

intense

medicinal chemistry effort made over the last 20 years has led to a variety of selective adenosine receptor agonists and antagonists. While all the agonists thus far identified are related to the adenosine structure, the antagonists available belong to different chemical

classes.

The prototypic antagonists are xanthine derivatives evolved from the natural compounds, caffeine and theophylline. Typically, they are 8-substituted-1,2,3-trialkylxanthine and are A1 selective antagonists. More recently, 8-styrylxanthines have been found to be selective for

A(2a)

receptors. Other non-xanthine heterocycles are potent A(2a) antagonists

not
of
and possess different degree of selectivity. Selective antagonists are available yet for A(2b) and A3 receptors. Given the recent developments

A(2a) selective antagonists, we have reviewed their chemical structures and biological properties in the attempts to get insight into this emerging class of new interesting compounds. The development of some of the A(2a) antagonists will provide better understanding of the role of A(2a) receptors in physiological and pathological states. The compounds appear to have the potential to be useful for the treatment of cerebral ischemia of neurodegenerative disorders, such as Parkinson's disease.

L11 ANSWER 8 OF 8 MEDLINE DUPLICATE 2
96401854 Document Number: 96401854. PubMed ID: 8991804. Cerebral ischemia

in gerbils: effects of acute and chronic treatment with adenosine A2A receptor agonist and antagonist. Von Lubitz D K; Lin R C; Jacobson K A. (Laboratory of Bioorganic Chemistry, NIH/NIDDK, Bethesda, MD 20892, USA.

) EUROPEAN JOURNAL OF PHARMACOLOGY, (1995 Dec 20) 287 (3) 295-302. Journal code: EN6; 1254354. ISSN: 0014-2999. Pub. country: Netherlands. Language: English.

AB Despite significant progress in understanding of the potential of adenosine A1 receptor-based therapies in treatment of cerebral ischemia and stroke, very little is known about the effect of selective stimulation

of adenosine A2A receptors on the outcome of a cerebrovascular arrest. In view of a major role played by adenosine A2 receptors in the regulation

of cerebral blood flow, we have investigated the effect of both acute and chronic administration of the selective adenosine receptor agonist 2-[(2-aminoethylamino)-carbonylphenylethylamino]-5'-N-ethylcarboxamido-adenosine (APEC) and antagonist 8-(3-chlorostyryl)caffeine (CSC) on the outcome of 10 min ischemia in gerbils. Acute treatment with APEC improved recovery of postischemic blood flow

and survival without affecting neuronal preservation in the hippocampus.

Acute treatment with CSC had no effect on the cerebral blood flow but resulted in a very significant protection of hippocampal neurons. Significant improvement of survival was present during the initial 10 days postischemia. Due to subsequent deaths of animals treated acutely with CSC, the end-point mortality (14 days postischemia) in this group did not differ statistically from that seen in the controls. It is, however, possible that the late mortality in the acute CSC group was caused by the systemic effects of brain ischemia that are not subject to the treatment with this drug. Chronic treatment with APEC resulted in a statistically significant improvement in all studied measures. Although chronic treatment with CSC improved postischemic blood flow, its effect on neuronal preservation was minimal and statistically insignificant. Mortality remained unaffected. The results indicate that the acute treatment with adenosine A2A receptor antagonists may have a limited

value in treatment of global ischemia. However, since administered CSC has no

effect on the reestablishment of postischemic blood flow, treatment of stroke with adenosine A2A receptor antagonists may not be advisable. Additional studies are necessary to elucidate whether chronically administered drugs acting at adenosine A2 receptors may be useful in treatment of stroke and other **neurodegenerative disorders**.

L12 1 FILE MEDLINE
L13 21 FILE CAPLUS
L14 16 FILE BIOSIS
L15 31 FILE EMBASE

TOTAL FOR ALL FILES

L16 69 (ALZHEIMER? OR PARKINSON?) AND L4

=> s l16 not l10

L17 1 FILE MEDLINE
L18 21 FILE CAPLUS
L19 14 FILE BIOSIS
L20 27 FILE EMBASE

TOTAL FOR ALL FILES

L21 63 L16 NOT L10

=> dup rem l21

PROCESSING COMPLETED FOR L21

L22 57 DUP REM L21 (6 DUPLICATES REMOVED)

=> d 1-57 cbib abs hitstr;select hit

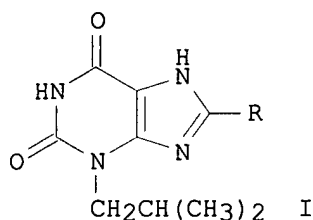
L22 ANSWER 1 OF 57 CAPLUS COPYRIGHT 2001 ACS

2001:167993 Document No. 134:212754 Selective antagonists of A2B adenosine receptors. Biaggioni, Italo O.; Feoktistov, Igor A.; Wells, Jack N. (Vanderbilt University, USA). PCT Int. Appl. WO 2001016134 A1 20010308, 24 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG,

BR,

BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 2000-US40751 20000828. PRIORITY: US 1999-PV151649 19990831.

GI

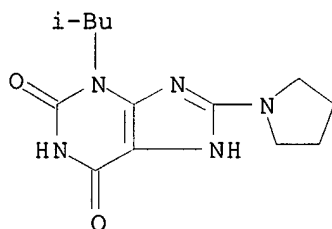


AB Xanthine derivs. of formula I (R = aliph. or cycloaliph. amine) or a pharmaceutically acceptable salt thereof are described as antagonists of A2B adenosine receptors. The compds. are formulated into tablets, aerosols, injections, and suppositories, and may be used to treat various diseases, including asthma and diarrhea.

IT **329024-77-3**
 RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (compns. and therapeutic uses of xanthine derivs. as selective antagonists of A2B adenosine receptors)

RN 329024-77-3 CAPLUS

CN 1H-Purine-2,6-dione, 3,7-dihydro-3-(2-methylpropyl)-8-(1-pyrrolidinyl)-
 (9CI) (CA INDEX NAME)



L22 ANSWER 2 OF 57 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
 2001200221 EMBASE KW-6002 Kyowa Hakko Kogyo. Knutsen L.J.S.; Weiss S.M..
 L.J.S. Knutsen, Vernalis Research Limited, Oakdene Court, 613 Reading
 Road, Winnersh, Wokingham, Berkshire, United Kingdom.
 L.Knutsen@vernalis.com. Current Opinion in Investigational Drugs 2/5
 (668-673) 2001.
 Refs: 38.
 ISSN: 0967-8298. CODEN: CIDREE. Pub. Country: United Kingdom. Language:
 English.

L22 ANSWER 3 OF 57 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
 2001173155 EMBASE Non-dopaminergic drug treatment of **Parkinson's**
 disease. Muller T.. T. Muller, Department of Neurology, St. Josef
 Hospital, Ruhr University Bochum, Gudrunstrasse 56, 44791 Bochum,
 Germany.
 thomas.mueller@ruhr-uni-bochum.de. Expert Opinion on Pharmacotherapy 2/4
 (557-572) 2001.

Refs: 127.

ISSN: 1465-6566. CODEN: EOPHF7. Pub. Country: United Kingdom. Language: English. Summary Language: English.

AB Several lines of evidence suggest that substitution of the dopaminergic striatal deficit only represents one important aspect of the treatment of **Parkinson's** disease (PD) because neurotransmitter systems other than the dopaminergic one also degenerate and aggravate **parkinsonian** motor, vegetative and cognitive symptoms. Thus, regulation and balance of altered non-dopaminergic neurotransmission could

provide an additional benefit for **parkinsonian** patients (PP). Moreover, onset of motor complications, psychosis and loss of drug efficacy increasingly reduce **parkinsonian** quality of life in the course of long-term dopamine substitution. Indirect stimulation of the dopaminergic neurotransmission via non-dopaminergic systems is an upcoming

interesting strategy to solve these problems. Treatment of L-dopa-associated dyskinesias represents a further important future task of non-dopaminergic drug therapy. NMDA antagonists are a promising therapeutic option but further trials are necessary to elucidate their efficacy. A further peripheral effect of L-dopa/dopa decarboxylase inhibitor (DDI) application is increased homocysteine synthesis with its putative hypothetical additional central impact on neurodegeneration and progression of PD. Long-term monitoring with subsequent therapeutic decrease of homocysteine levels with folic acid could result in substantial clinical benefits at reasonable costs for PP. Also, it could hypothetically influence altered dopaminergic and non-dopaminergic neurotransmission beside its impact on occurrence of vascular disease and altered striatal microvascularisation in PD. The interesting field of non-dopaminergic drug therapy is emerging and will hopefully lead to a better understanding of PD and subsequently improve drug therapy of **parkinsonian** symptoms, which do not respond to dopaminergic substitution or are long-term complications of dopamine substitution.

L22 ANSWER 4 OF 57 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.

2001161428 EMBASE Adenosine A(2A) receptor enhances GABA(A)-mediated IPSCs in

the rat globus pallidus. Shindon T.; Mori A.; Kase H.; Ichimura M.. M. Ichimura, Pharmaceutical Research Institute, Kyowa Hakko Kogyo Co. Ltd., 1188 Shimotogari Nagaizumi, Sunto, Shizuoka 411-8731, Japan. michio.ichimura@kyowa.co.jp. Journal of Physiology 532/2 (423-434) 15 Apr 2001.

Refs: 50.

ISSN: 0022-3751. CODEN: JPHYA7. Pub. Country: United Kingdom. Language: English. Summary Language: English.

AB 1. The actions of adenosine A(2A) receptor agonists were examined on GABAergic synaptic transmission in the globus pallidus (GP) in rat brain slices using whole-cell patch-clamp recording. GP neurones were characterized into two major groups, type I and type II, according to the degree of time-dependent hyperpolarization-activated inward rectification and the size of input resistance. 2. The A(2A) receptor agonist

(CGS21680;

0.3-3 .mu.M) enhanced IPSCs evoked by stimulation within the GP. The actions of CGS21680 were blocked by the A(2A) antagonists (E)-8-(3,4-dimethoxystyryl)-1,3-dipropyl-7-methylxanthine (KF17837) and

4-(2-[7-amino-2-(2-fury)[1,2,4]triazolo[2,3-a]-[1,3,5]triazin-5-ylamino]ethyl) pheno (ZM241385). [[3. The CGS21680-induced increase in IPSCs was associated with a reduction in paired-pulse facilitation. CGS21680 (0.3 .mu.M) increased the frequency of miniature IPSCs (mIPSCs) without affecting mIPSC amplitude. These observations demonstrated that the enhancement of IPSCs in the GP was attributable to presynaptic, but not postsynaptic, A(2A) receptors. [[4. The results suggest that A(2A) receptors in the GP serve to inhibit GP neuronal activity, thereby disinhibiting subthalamic nucleus neurone activity. Thus, the A(2A) receptor-mediated presynaptic regulation in the GP, together with the A(2A) receptor-mediated intrastriatal presynaptic control of GABAergic neurotransmission described previously, may play a crucial role in controlling the neuronal functions of basal ganglia. This A(2A) receptor-mediated presynaptic dual control in the striatopallidal pathway could also afford the mode of action of A(2A) antagonists for

ameliorating

the symptoms of **Parkinson's** disease in an animal model.

L22 ANSWER 5 OF 57 BIOSIS COPYRIGHT 2001 BIOSIS

2001:262667 Document No.: PREV200100262667. The A2A adenosine receptor antagonists are effective in models of **Parkinson's** disease through the modulation of both D2 and D1 dopaminergic pathways. Bastia,

E.

(1); Impagnatiello, F. (1); Fredduzzi, S. (1); Ongini, E. (1); Monopoli, A. (1). (1) Schering-Plough Research Institute, San Raffaele Science

Park,

Via Olgettina, 58, 20132, Milan Italy. British Journal of Pharmacology, (May, 2001) Vol. 133, No. Proceedings Supplement, pp. 160P. print.

Meeting

Info.: Proceedings of the British Pharmacological Society Meeting Birmingham, UK December 18-21, 2000 British Pharmacological Society.

ISSN:

0007-1188. Language: English. Summary Language: English.

L22 ANSWER 6 OF 57 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 1

2001:215926 Document No.: PREV200100215926. KW-6002: Antiparkinsonian, antidepressant, adenosine A2A antagonist. Rabasseda, X. (1); Sorbera, L. A. (1); Martin, L. (1); Leeson, P. A. (1); Castaner, J. (1). (1) Prous Science, 08080, Barcelona Spain. Drugs of the Future, (January, 2001)

Vol.

26, No. 1, pp. 20-24. print. ISSN: 0377-8282. Language: English. Summary Language: English.

L22 ANSWER 7 OF 57 CAPLUS COPYRIGHT 2001 ACS

2000:525694 Document No. 133:217584 Rescue of locomotor impairment in dopamine D2 receptor-deficient mice by an adenosine A2A receptor antagonist. Aoyama, Shiro; Kase, Hiroshi; Borrelli, Emiliana (Institut

de

Genetique et de Biologie Moleculaire et Cellulaire, C.U. de Strasbourg, Fr.). J. Neurosci., 20(15), 5848-5852 (English) 2000. CODEN: JNRSDS. ISSN: 0270-6474. Publisher: Society for Neuroscience.

AB

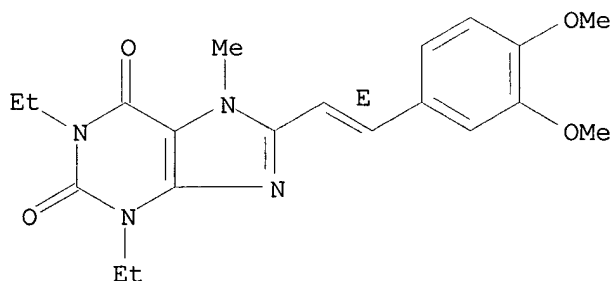
In **Parkinson's** disease a degeneration of dopaminergic neurons of the nigrostriatal pathway is obsd. Loss of dopaminergic regulation of striatal neuron activity results in altered motor functions. Adenosine A2A (A2AR) and dopamine D2 (D2R) receptors are colocalized in striatal

medium spiny neurons. It has been proposed that adenosine binding to A2AR lowers the affinity of dopamine for D2R, thus modulating the function of this receptor. Absence of D2R in knockout mice (D2R^{-/-}) results in impaired locomotion and coordinated movements. This indicates that absence of dopamine in **Parkinson's** disease might principally affect D2R-mediated effects with regard to locomotor functions. A2AR-selective antagonists have been demonstrated to have anti-**parkinsonian** activities in various models of **Parkinson's** disease in rodents and nonhuman primates. In this article, D2R^{-/-} mice were used to explore the possibility that an A2AR antagonist might reestablish their motor impairment. Interestingly, blockade of A2AR rescues the behavioral parameters altered in D2R^{-/-} mice. In addn., the level of expression of enkephalin and substance P, which were altered in D2R^{-/-}, were also reestablished to normal levels after A2AR antagonist treatment. These results show that A2AR and D2R have antagonistic and independent activities in controlling neuronal and motor functions in the basal ganglia. They also provide evidence that selective A2AR antagonists can exhibit their anti-**parkinsonian** activities through a nondopaminergic mechanism.

IT 155270-99-8, KW 6002
 RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
 (rescue of locomotor impairment in dopamine D2 receptor-deficient mice by adenosine A2A receptor antagonist)

RN 155270-99-8 CAPLUS
 CN 1H-Purine-2,6-dione, 8-[(1E)-2-(3,4-dimethoxyphenyl)ethenyl]-1,3-diethyl-3,7-dihydro-7-methyl- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



L22 ANSWER 8 OF 57 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
 2000105439 EMBASE Purinergic and pyrimidinergic receptors as potential drug targets. Williams M.; Jarvis M.F.. Dr. M. Williams, Neurological/Urological Dis. Res., Abbott Laboratories, 100 Abbott Park Road, Abbott Park, IL 60064-6125, United States.
 mike.williams@abbott.com.
 Biochemical Pharmacology 59/10 (1173-1185) 15 May 2000.
 Refs: 109.
 ISSN: 0006-2952. CODEN: BCPCA6.
 Publisher Ident.: S 0006-2952(99)00341-X. Pub. Country: United States.

Language: English. Summary Language: English.

AB In the last decade, the field of purinergic pharmacology has continued to grow as the complexity of the receptor families and the various enzymes involved in purine metabolism have been defined in molecular terms. A major theme that has emerged from these studies is the functional complexity of the interactions between P1 and P2 receptors, based upon the dynamic interrelationship between ATP and adenosine as extracellular signaling molecules. It is now clear that ATP and its degradation products (particularly ADP and adenosine) form a complex cascade for the regulation of cell-to-cell communication that can function to attenuate the consequences of tissue trauma (e.g. ischemia) that involve alterations in cellular energy charge and depletion of ATP stores. In addition to the P2 receptor family, alterations in cellular ATP stores can also affect the function of other receptors, e.g. K(ATP) channels, and mitochondrial function. The discovery of pyrimidine-preferring (UTP/UDP) P2Y receptors has also raised the possibility that the corresponding nucleoside, uracil, may function as a signaling molecule. Copyright (C) 2000 Elsevier Science Inc.

L22 ANSWER 9 OF 57 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.

2000439043 EMBASE A(2A) adenosine receptor antagonists - Future drugs for **Parkinson's** disease?. Muller C.E.. C.E. Muller, University of Bonn, Pharmaceutical Institute, Pharmaceutical Chemistry Poppelsdorf, Kreuzbergweg 26, D-53115 Bonn, Germany. Drugs of the Future 25/10 (1043-1052) 2000.

Refs: 101.

ISSN: 0377-8282. CODEN: DRFUD4. Pub. Country: Spain. Language: English.

L22 ANSWER 10 OF 57 CAPLUS COPYRIGHT 2001 ACS

2000:201928 Document No. 133:202924 Combined Use of the Adenosine A2A Antagonist KW-6002 with l-DOPA or with Selective D1 or D2 Dopamine Agonists Increases Antiparkinsonian Activity but Not Dyskinesia in MPTP-Treated Monkeys. Kanda, Tomoyuki; Jackson, Michael J.; Smith, Lance A.; Pearce, Ronald K. B.; Nakamura, Joji; Kase, Hiroshi; Kuwana, Yoshihisa; Jenner, Peter (Pharmaceutical Research Institute, Kyowa Hakko Kogyo Co., Ltd., Shizuoka, 411-8731, Japan). Exp. Neurol., 162(2), 321-327 (English) 2000. CODEN: EXNEAC. ISSN: 0014-4886. Publisher: Academic Press.

AB The novel selective adenosine A2A receptor antagonist KW-6002 improves motor disability in MPTP-treated **parkinsonian** marmosets without provoking dyskinesia. In this study we have investigated whether KW-6002 in combination with l-DOPA or selective D1 or D2 dopamine receptor agonists enhances antiparkinsonian activity in MPTP-treated common marmosets. Combination of KW-6002 with the selective dopamine D2 receptor agonist quinpirole or the D1 receptor agonist SKF80723 produced an additive improvement in motor disability. Coadministration of KW-6002 with a low dose of l-DOPA also produced an additive improvement in motor disability, and increased locomotor activity. The ability of KW-6002 to enhance antiparkinsonian activity was more marked with l-DOPA and quinpirole than with the D1 agonist. However, despite producing an

enhanced antiparkinsonian response KW-6002 did not exacerbate l-DOPA-induced dyskinesia in MPTP-treated common marmosets previously primed to exhibit dyskinesia by prior exposure to l-DOPA. Selective adenosine A2A receptor antagonists, such as KW-6002, may be one means of reducing the dosage of l-DOPA used in treating **Parkinson's** disease and are potentially a novel approach to treating the illness both as monotherapy and in combination with dopaminergic drugs. (c) 2000 Academic Press.

IT 155270-99-8, KW-6002

RL: BAC (Biological activity or effector, except adverse); THU

(Therapeutic use); BIOL (Biological study); USES (Uses)

(adenosine A2A antagonist KW-6002 combination with L-DOPA or D1/D2

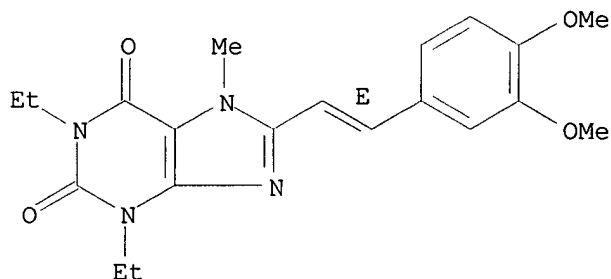
dopamine agonists increases antiparkinsonian action but not

dyskinesia)

RN 155270-99-8 CAPLUS

CN 1H-Purine-2,6-dione, 8-[(1E)-2-(3,4-dimethoxyphenyl)ethenyl]-1,3-diethyl-3,7-dihydro-7-methyl- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



L22 ANSWER 11 OF 57 CAPLUS COPYRIGHT 2001 ACS

DUPLICATE 2

2000:828333 Document No. 134:157476 Adenosine A2A receptor antagonists KF17837 and KW-6002 potentiate rotation induced by dopaminergic drugs in hemi-**Parkinsonian** rats. Koga, K.; Kurokawa, M.; Ochi, M.; Nakamura, J.; Kuwana, Y. (Pharmaceutical Research Institute, Kyowa Hakko Kogyo Co. Ltd., Shizuoka, Sunto-gun, Nagaizumi-cho, 411-8731, Japan). Eur. J. Pharmacol., 408(3), 249-255 (English) 2000. CODEN: EJPHAZ.

ISSN:

0014-2999. Publisher: Elsevier Science B.V..

AB The effects of novel adenosine A2A receptor antagonists KF17837 ((E)-1,3-dipropyl-8-(3,4-dimethoxystyryl)-7-methyl-3,7-dihydro-1H-purine-2,6-dione) and KW-6002 ((E)-1,3-diethyl-8-(3,4-dimethoxystyryl)-7-methyl-3,7-dihydro-1H-purine-2,6-dione), on rotational behavior induced by apomorphine or L-DOPA (l-3,4-dihydroxyphenylalanine) were investigated in rats with unilateral 6-hydroxydopamine lesions. Both KF17837 and KW-6002 slightly induced rotational behavior per se. However, KF17837 and

KW-6002

significantly increased the total counts of turning induced by apomorphine

at doses of 3 mg/kg, p.o. and 10 mg/kg, p.o., and at doses of 1 mg/kg, p.o. and higher, resp. KF17837 and KW-6002 also potentiated the rotational behavior induced by L-DOPA at a dose of 3 mg/kg, p.o.

Furthermore, i.c.v. injection (10 .mu.g/20 .mu.l) of a selective adenosine

A2 receptor agonist CGS 21680 {2-[p-(2-carboxyethyl)phenethylamino]-5'-N-ethylcarboxamidoadenosine} partially prevented the rotational behavior induced by apomorphine and this inhibition was reversed by KW-6002 (1 mg/kg, p.o.). The increase in total counts of apomorphine-induced turning

by the adenosine A2A receptor antagonists seems to be mainly attributable to prolongation of turning duration rather than enhancement of intensity. These results suggest that these adenosine A2A receptor antagonists may

be useful to ameliorate shortening in the duration of dopaminergic drug response in patients with advanced **Parkinson's** disease.

IT 141807-96-7, KF17837 155270-99-8, KW-6002

RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

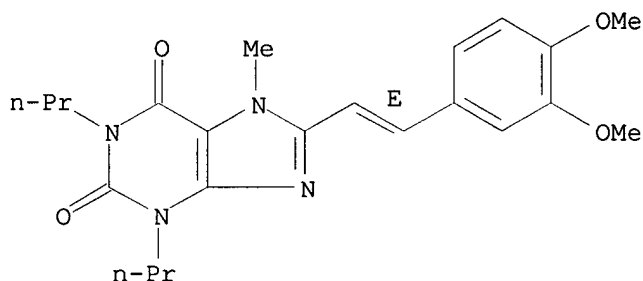
(adenosine A2A receptor antagonists KF17837 and KW-6002 potentiate rotation induced by dopaminergic drugs in hemi-**Parkinsonian** rats)

RN 141807-96-7 CAPLUS

CN 1H-Purine-2,6-dione,

8-[(1E)-2-(3,4-dimethoxyphenyl)ethenyl]-3,7-dihydro-7-methyl-1,3-dipropyl- (9CI) (CA INDEX NAME)

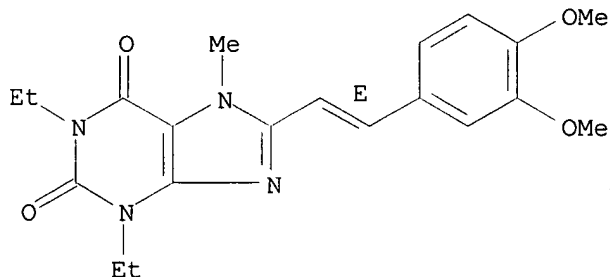
Double bond geometry as shown.



RN 155270-99-8 CAPLUS

CN 1H-Purine-2,6-dione, 8-[(1E)-2-(3,4-dimethoxyphenyl)ethenyl]-1,3-diethyl-3,7-dihydro-7-methyl- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



L22 ANSWER 12 OF 57 CAPLUS COPYRIGHT 2001 ACS

2000:71207 Document No. 133:587 Selective adenosine A2A receptor antagonism as an alternative therapy for **Parkinson's** disease: a study in nonhuman primates. Tahar, A. Hadj; Grondin, R.; Gregoire, L.; Bedard, P. J.; Mori, A.; Kase, H. (Neuroscience Research Unit, Laval University Research Center, Ste-Foy, PQ, Can.). Adenosine Recept. Parkinson's Dis., 229-244. Editor(s): Kase, Hiroshi; Richardson, Peter J.; Jenner, Peter. Academic Press: San Diego, Calif. (English) 2000. CODEN: 68PKAX.

AB The aim of the study was to evaluate , in MPTP exposed monkeys having a stable **parkinsonian** syndrome and exhibiting dyskinesias to levodopa, both the antiparkinsonian and diskinetetic effects upon challenge with the selective adenosine A2A receptor antagonist KW-6002. The results

showed that KW-6002 administered alone increased locomotion and improved **parkinsonian** symptoms in a significant manner in MPTP-treated monkeys. Coadministration of KW-6002 with L-DOPA, increased L-DOPA response. (c) 2000 Academic Press.

IT 155270-99-8, KW 6002

RL: BAC (Biological activity or effector, except adverse); BPR

(Biological

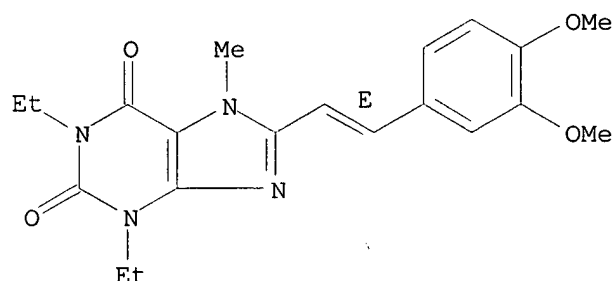
process); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)

(adenosine receptor antagonism as an alternative therapy for **Parkinson's** disease)

RN 155270-99-8 CAPLUS

CN 1H-Purine-2,6-dione, 8-[(1E)-2-(3,4-dimethoxyphenyl)ethenyl]-1,3-diethyl-3,7-dihydro-7-methyl- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



L22 ANSWER 13 OF 57 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.

2000368904 EMBASE Adenosine receptors and **Parkinson's** disease.

Kostic V.S.; Przedborski S.. V.S. Kostic, Institute of Neurology CCS, Belgrade, Yugoslavia. Focus on Parkinson's Disease 12/3 (60-62) 2000. Refs: 36.

ISSN: 0924-2015. CODEN: FPDIF2. Pub. Country: Netherlands. Language: English. Summary Language: English.

AB Levodopa is the most effective symptomatic treatment for **Parkinson's** disease (PD). Chronic use of levodopa is, however, frequently complicated by the development of disabling motor side effects (motor

response fluctuations and dyskinesia) that can limit this pharmacological approach [1]; these side effects may result from alterations in the finely-tuned neurochemical balance in the output pathways of basal ganglia. Thus, attention has turned toward nondopaminergic targets on the basal-ganglia neural pathways as an alternative means of treating PD. An ideal therapeutic target is the adenosine A(2A) receptor, which is located together with the dopamine (DA) D2 receptor on neurons of the indirect striatopallidal pathway [2].

L22 ANSWER 14 OF 57 BIOSIS COPYRIGHT 2001 BIOSIS

2001:88847 Document No.: PREV200100088847. Adenosine A2A receptor enhances GABAA-mediated IPSCs in the globus pallidus. Shindoh, T. (1); Mori, A.; Kase, H.; Ichimura, M.. (1) Kyowa Hakko Kogyo Co Ltd, Shizuoka-Ken Japan. Society for Neuroscience Abstracts, (2000) Vol. 26, No. 1-2, pp. Abstract No.-431.18. print. Meeting Info.: 30th Annual Meeting of the Society of Neuroscience New Orleans, LA, USA November 04-09, 2000 Society for Neuroscience. ISSN: 0190-5295. Language: English. Summary Language: English.

AB We found that the adenosine A2A receptor serves to enhance the GABAergic synaptic transmission in the globus pallidus (GP) in rat GP slices using whole-cell patch-clamp recording. The A2A receptor agonist, CGS21680 (0.3-3 μ M) enhanced IPSCs evoked by stimulation within the GP. The actions of CGS21680 were blocked by A2A antagonists, KF17837 or ZM241385. This CGS21680-induced enhancement was associated with a reduction in paired-pulse facilitation. CGS21680 increased the frequency of miniature IPSCs (mIPSCs) without affecting mIPSC amplitude. These observations demonstrated that the enhancement of IPSCs in the GP was attributable to presynaptic, but not postsynaptic, A2A receptors. We previously reported that presynaptic A2A receptors serve to suppress the intrastriatal GABAergic synaptic transmission. This striatal A2A receptor-mediated disinhibition could cause the overactivity for the striatal GABAergic output and thereby exert an inhibitory action on GP neurons. Combined

with the result in this study, GP neurons could be regulated to suppression via

both striatal and pallidal A2A receptors. This A2A receptor-mediated inhibition for GP neurons might afford very effective control over the GP/subthalamus circuitry. Thus, these A2A receptor-mediated regulations may play an crucial role in the basal ganglia, which could explain the mode of action of A2A antagonists for ameliorating symptoms in the **Parkinson's** disease model.

L22 ANSWER 15 OF 57 BIOSIS COPYRIGHT 2001 BIOSIS

2001:89843 Document No.: PREV200100089843. Novel neuroprotection by A2A adenosine receptor inactivation in the MPTP model of **Parkinson's** disease. Chen, J. F. (1); Staal, R.; Xu, K.; Beilstein, M.; Sonsalla, P. K.; Schwarzschild, M. A.. (1) Mass. Gen. Hospital " Harvard Med. Sch., Charlestown, MA USA. Society for Neuroscience Abstracts, (2000) Vol. 26, No. 1-2, pp. Abstract No.-478.1. print. Meeting Info.: 30th Annual

Meeting of the Society of Neuroscience New Orleans, LA, USA November 04-09, 2000 Society for Neuroscience. ISSN: 0190-5295. Language: English. Summary Language: English.

AB A2A Adenosine receptors are highly enriched in the striatum where they

modulate dopaminergic motor activity. A2A receptor antagonists are now entering human trials for **Parkinson's** disease (PD) based on their motor enhancement feature in animal models of PD. However, the effects of A2A receptor blockade on the neurodegeneration underlying PD

is

entirely unknown. In this study, complementary genetic and pharmacological

approaches to A2A receptor inactivation reveal a novel neuroprotection in the MPTP model of PD. MPTP-induced depletion of dopamine and dopamine transporter levels was significantly attenuated in the striatum of A2A receptor knockout (A2A KO) mice and of mice pretreated with A2A

antagonist

8-(3-chlorostyryl)caffeine (CSC, 5 mg/kg). The extent of striatal dopamine

depletion was however indistinguishable between A2A KO and wild-type mice in response to MPP+, the active metabolite of MPTP, implicating proximal events in the cascade of MPTP toxicity. Analysis of striatal MPTP metabolites after intraperitoneal MPTP administration revealed elevated levels of MPTP, but diminished levels of its oxidation products MPDP+ and MPP+ in A2A KO or CSC-treated mice. The data suggest that inhibition of monoamine oxidase activity contributes to the reduction of MPP+ levels and, in turn, the attenuation of neurotoxicity. Furthermore, the previously described motor stimulant effect of CSC acting as an A2A specific antagonist was confirmed by its demonstration in wild-type but not A2A KO mice. Together, the novel neuroprotective effects of A2A receptor inactivation, coupled with the established short-term motor benefits of A2A receptor antagonists, significantly enhance the therapeutic potential of these agents for the treatment of PD.

L22 ANSWER 16 OF 57 BIOSIS COPYRIGHT 2001 BIOSIS

1999:412016 Document No.: PREV199900412016. Antiparkinsonian activity of adenosine A2A antagonists in experimental models. Kuwana, Yoshihisa (1); Shiozaki, S. (1); Kanda, T. (1); Kurokawa, M. (1); Koga, K. (1); Ochi, M. (1); Ikeda, K. (1); Kase, Hiroshi (1); Jackson, Michael John; Smith,

Lance

A.; Pearce, R. K. B.; Jenner, Peter George. (1) Pharmaceutical Research Institute, Kyowa Hakko Kogyo Company, Shizuoka, 411 Japan. Stern, G. M. [Editor]. Advances in Neurology, (1999) Vol. 80, pp. 121-123. Advances in Neurology; Parkinson's disease. Publisher: Lippincott Williams and

Wilkins

227 East Washington Square, Philadelphia, Pennsylvania 19106, USA.

Meeting

Info.: Selected Papers from the Twelvth International Symposium on Parkinson's Disease ISSN: 0091-3952. ISBN: 0-7817-1598-9. Language: English.

L22 ANSWER 17 OF 57 BIOSIS COPYRIGHT 2001 BIOSIS

1999:412015 Document No.: PREV199900412015. Adenosine receptor antagonists and

Parkinson's disease: Actions of the A2A receptor in the striatum. Richardson, Peter J. (1); Gubitz, Amelie K. (1); Freeman, Tom C.; Dixon, Alistair K.. (1) Department of Pharmacology, University of Cambridge, Cambridge, CB2 1QJ UK. Stern, G. M. [Editor]. Advances in Neurology, (1999) Vol. 80, pp. 111-119. Advances in Neurology; Parkinson's disease. Publisher: Lippincott Williams and Wilkins 227 East Washington Square,

Philadelphia, Pennsylvania 19106, USA. Meeting Info.: Selected Papers
from
the Twelvth International Symposium on Parkinson's Disease ISSN:
0091-3952. ISBN: 0-7817-1598-9. Language: English.

L22 ANSWER 18 OF 57 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
1999341033 EMBASE What is new in degenerative dementia disorders?. Jellinger
K.A.. Dr. K.A. Jellinger, Ludwig-Boltzmann-Inst. Klin. Neuro.,
Baumgartner

Hohe 1, A-1140 Wien, Austria. kurt.jellinger@univie.ac.at. Wiener
Klinische Wochenschrift 111/17 (682-704) 17 Sep 1999.
Refs: 217.

ISSN: 0043-5325. CODEN: WKWOAO. Pub. Country: Austria. Language: English.
Summary Language: English; German.

AB **Alzheimer's** disease and other degenerative disorders - dementia
with Lewy bodies, frontotemporal dementia, etc. - causing about 90% of
dementias in advanced age, are a major health problem of increasing
practical, scientific, and socio-economic importance. Despite
considerable
progress in genetic, clinical and basic neurosciences, the aetiology and
molecular mechanisms of these disorders are still unknown and their early
diagnosis, due to lack of specific biomarkers, is still unsatisfactory.
The epidemiology, risk factors, clinical and morphological diagnostic
criteria, probable pathogenic factors, and molecular genetics of the

major
types of degenerative dementias are reviewed. Their management involves
several pharmacologic, non-pharmacologic and psychosocial options.

Modification of the disease by reducing known and presumable risk
factors,
cognitive enhancement with cholinomimetic drugs, and reduction of
behavioural abnormalities with psychotropic drugs, together with informed
community and private management are currently achievable goals that will
serve to delay the progression of disease. In the future, these options
will hopefully be replaced by more effective management strategies in
order to improve the quality of life of both, patients and caregivers.

L22 ANSWER 19 OF 57 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
1999428786 EMBASE [New perspectives in the treatment of the motor
complications associated with the chronic treatment of **Parkinson**
disease]. NUEVAS PERSPECTIVAS EN EL TRATAMIENTO DE LAS COMPLICACIONES
MOTORAS ASOCIADAS AL TRATAMIENTO CRONICO DE LA ENFERMEDAD DE
PARKINSON. Linazasoro G.. Dr. G. Linazasoro, Centro
Neurol./Neurocir. Funcional, Clinica Quiron, Parque de Alcolea, s/n,
20012
San Sebastian, Spain. Neurotax@jet.es. Neurologia 14/8 (393-406) 1999.
Refs: 98.
ISSN: 0213-4853. CODEN: NERLEN. Pub. Country: Spain. Language: Spanish.

L22 ANSWER 20 OF 57 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
1999262411 EMBASE Distribution, biochemistry and function of striatal
adenosine A(2A) receptors. Svenningsson P.; Le Moine C.; Fisone G.;
Fredholm B.B.. B.B. Fredholm, Section Molecular Neuropharmacology,
Department Physiology/Pharmacology, Karolinska Institutet, 171 77
Stockholm, Sweden. bertil.fredholm@fyfa.ki.se. Progress in Neurobiology
59/4 (355-396) 1999.

Refs: 442.

ISSN: 0301-0082. CODEN: PGNBA5.

Publisher Ident.: S 0301-0082(99)00011-8. Pub. Country: United Kingdom.

Language: English. Summary Language: English.

AB It is well known that the nucleoside adenosine exerts a modulatory influence in the central nervous system by activating G protein coupled receptors. Adenosine A(2A) receptors, the subject of the present review, are predominantly expressed in striatum, the major area of the basal ganglia. Activation of A(2A) receptors interferes with effects mediated by most of the principal neurotransmitters in striatum. In particular, the inhibitory interactions between adenosine acting on A(2A) receptors and dopamine acting on D2 receptors have been well examined and there is much evidence that A(2A) receptors may be a possible target for future development of drugs for treatment of **Parkinson's** disease, schizophrenia and affective disorders. Our understanding of the role of striatal A(2A) receptors has increased dramatically over the last few years. New selective antibodies, antagonist radioligands and optimized in situ hybridization protocols have provided detailed information on the distribution of A(2A) receptors in rodent as well as primate striatum. Studies on the involvement of A(2A) receptors in the regulation of DARPP-32 and the expression of immediate early genes, such as nerve growth factor-induced clone A and c-fos, have pointed out an important role for these receptors in regulating striatopallidal neurotransmission. Moreover, by using novel selective antagonists for

A(2A) receptors and transgenic mice lacking functional A(2A) receptors, crucial information on the behavioral role of striatal A(2A) receptors has been provided, especially concerning their involvement in the stimulatory action of caffeine and the anti-**Parkinsonian** properties of A(2A) receptor antagonists. In the present review, current knowledge on the distribution, biochemistry and function of striatal A(2A) receptors is summarized. Copyright (C) 1999 Elsevier Science Ltd.

L22 ANSWER 21 OF 57 BIOSIS COPYRIGHT 2001 BIOSIS
1999:538599 Document No.: PREV199900538599. Radiosynthesis and biodistribution

of (11C)LZ66, a new potent and selective ligand for A2A receptor system. Todde, S. (1); Moresco, R. M. (1); Monopoli, A.; Simonelli, P. (1); Cacciari, B.; Baraldi, P.; Matarrese, M. (1); Carpinelli, A. (1); Kienle, M. Galli (1); Fazio, F. (1). (1) CNR-INB, Scientific Institute H San Raffaele, University of Milan, Via Olgettina 60, I-20132, Milan Italy. Journal of Labelled Compounds and Radiopharmaceuticals, (June, 1999) Vol. 42, No. SUPPL. 1, pp. S176-S178. Meeting Info.: XIIIth International Symposium on Radiopharmaceutical Chemistry St. Louis, Missouri, USA June 27-July 1, 1999 ISSN: 0362-4803. Language: English.

L22 ANSWER 22 OF 57 CAPLUS COPYRIGHT 2001 ACS
2000:97711 Document No. 132:260554 Actions of adenosine A2A receptor antagonist KW-6002 on drug-induced catalepsy and hypokinesia caused by reserpine or MPTP. Shiozaki, Shizuo; Ichikawa, Shunji; Nakamura, Joji; Kitamura, Shigeto; Yamada, Koji; Kuwana, Yoshihisa (Pharmaceutical Research Institute, Kyowa Hakko Kogyo Co. Ltd, Shizuoka, 411-8731, Japan). Psychopharmacology (Berlin), 147(1), 90-95 (English) 1999. CODEN: PSCHDL.

ISSN: 0033-3158. Publisher: Springer-Verlag.

AB Rationale: Current treatment of **Parkinson's** disease (PD) is based on dopamine replacement therapy, but this leads to long term complications, including dyskinesia. Adenosine A2A receptors are particularly abundant in the striatum and would be a target for an alternative approach to the treatment of PD. Objectives: The purpose of this study is to examine the efficacy and potency of the novel selective adenosine A2A receptor antagonist

(E)-1,3-diethyl-8-(3,4-dimethoxystyryl)-7-methyl-3,7-dihydro-1H-purine-2,6-dione (KW-6002) in ameliorating the motor deficits in various mouse models of **Parkinson's** disease. Methods: We evaluated the efficacy and potency of KW-6002 and other ref. compds. in the selective adenosine A2A receptor agonist 2-[p-(2-carboxyethyl)phenethylamino]-5'-N-ethylcarboxamidoadenosine (CGS 21680)-, haloperidol- or reserpine-induced catalepsy models. The effect of KW-6002 on reserpine or 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine hydrochloride (MPTP)-induced hypolocomotion was also examd. Results: The ED50s of KW-6002 in the reversal of CGS 21680-induced and reserpine-induced catalepsy were 0.05 mg/kg, PO and 0.26 mg/kg, PO, resp. Compared to the ED50 of other adenosine antagonists and dopamine agonist drugs, KW-6002 is over 10 times as potent in these models. KW-6002 also ameliorated the hypolocomotion (min. ED; 0.16 mg/kg) induced by nigral dopaminergic dysfunction with MPTP or reserpine treatment. Combined administrations of subthreshold doses of KW-6002 and L-dopa (50 mg/kg,

PO) exerted prominent effects on haloperidol-induced and reserpine-induced catalepsy, suggesting that there may be a synergism between the adenosine A2A receptor antagonist KW-6002 and dopaminergic agents. Conclusions: To our knowledge, KW-6002 is the most potent and orally active adenosine A2A receptor antagonist in exptl. models of **Parkinson's** disease, and may offer a new therapeutic approach to the treatment of **Parkinson's** disease.

IT 155270-99-8, KW 6002

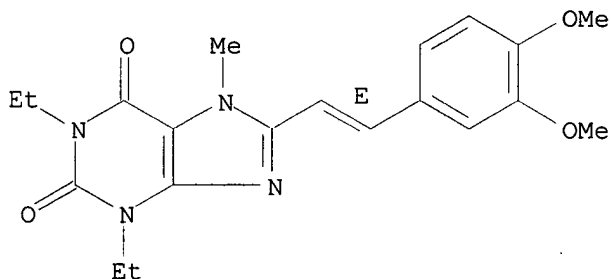
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(actions of adenosine A2A receptor antagonist KW-6002 on drug-induced catalepsy and hypokinesia caused by reserpine or MPTP)

RN 155270-99-8 CAPLUS

CN 1H-Purine-2,6-dione, 8-[(1E)-2-(3,4-dimethoxyphenyl)ethenyl]-1,3-diethyl-3,7-dihydro-7-methyl- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



L22 ANSWER 23 OF 57 CAPLUS COPYRIGHT 2001 ACS

1998:644563 Document No. 130:33316 Adenosine A2A receptors modify motor function in MPTP-treated common marmosets. Kanda, Tomoyuki; Tashiro, Tomomi; Kuwana, Yoshihisa; Jenner, Peter (Pharmaceutical Research Institute, Kyowa Hakko Kogyo Co Ltd, Shizuoka, 411-8731, Japan). NeuroReport, 9(12), 2857-2860 (English) 1998. CODEN: NERPEZ. ISSN: 0959-4965. Publisher: Lippincott Williams & Wilkins.

AB Both adenosine A1 and A2 receptor populations are located in the striatum and can modify locomotor activity, and they may form a therapeutic target for **Parkinson's** disease (PD). Administration of the selective adenosine A2A antagonist

(E)-1,3-diethyl-8-(3,4-dimethoxystyryl)-7-methyl-3,7-dihydro-1H-purine-2,6-dione (KW-6002) to MPTP-treated common marmosets

increased locomotor activity. In contrast, administration of the selective A1 receptor antagonist 1,3-dipropyl-8-cyclopentylxanthine (DPCPX)

had no effect on locomotion. Administration of the adenosine A2A receptor

agonist 2-[p-[2-(2-aminoethylamino) carbonyl-ethyl] phenethyl amino]-5'-N-ethylcarboxamidoadenosine (APEC) dose dependently suppressed basal locomotor activity. A minimally ED of APEC (0.62 mg/kg, i.p) completely reversed the increase in locomotor activity produced by administration of KW-6002. The adenosine A2A receptor appears to be an important target for the treatment of basal ganglia disorders, particularly PD.

IT 155270-99-8

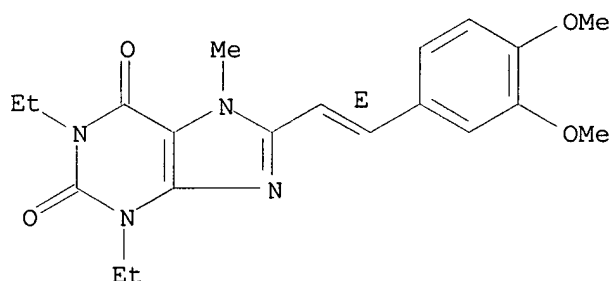
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(adenosine A2A receptors modify motor function in MPTP-treated common marmoset **Parkinsonism** model)

RN 155270-99-8 CAPLUS

CN 1H-Purine-2,6-dione, 8-[(1E)-2-(3,4-dimethoxyphenyl)ethenyl]-1,3-diethyl-3,7-dihydro-7-methyl- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



L22 ANSWER 24 OF 57 CAPLUS COPYRIGHT 2001 ACS

1998:418387 Document No. 129:198300 Motor effects induced by a blockade of adenosine A2A receptors in the caudate-putamen. Hauber, W.; Nagel, J.; Sauer, R.; Muller, C. E. (Department of Animal Physiology, Institute of

Biology, University of Stuttgart, Stuttgart, D-70550, Germany).
NeuroReport, 9(8), 1803-1806 (English) 1998. CODEN: NERPEZ. ISSN:
0959-4965. Publisher: Rapid Science Publishers.

AB Motor effects mediated through adenosine A2A receptors within the caudate-putamen were investigated in rats using bilateral microinfusions of MSX-3 (9 .mu.g in 1 .mu.l per side), a water-sol. phosphate prodrug of the selective A2A receptor antagonist MSX-2. Blockade of striatal A2A receptors produced a significant motor stimulation measured by an enhanced

D1 sniffing activity. Furthermore, catalepsy induced by systemic dopamine (0.75 mg/kg SCH23390, i.p.) or dopamine D2 receptor blockade (1.5 mg/kg raclopride, i.p.) was potently reversed. These findings suggest that A2A receptors within the caudate-putamen are tonically activated by

endogenous adenosine and that a striatal A2A receptor blockade produces motor stimulant effects, in particular in animals with dopamine hypofunction. The present results support the view that A2A receptor antagonists may be potentially useful therapeutics for the treatment of **Parkinson's** disease.

IT 212131-38-9

RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)

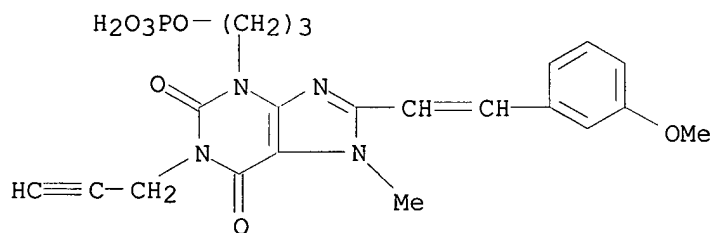
(motor effects induced by blockade of adenosine A2A receptors in caudate-putamen of rats)

RN 212131-38-9 CAPLUS

CN 1H-Purine-2,6-dione,

3,7-dihydro-8-[2-(3-methoxyphenyl)ethenyl]-7-methyl-3-

[3-(phosphonoxy)propyl]-1-(2-propynyl)-, disodium salt (9CI) (CA INDEX NAME)



●2 Na

L22 ANSWER 25 OF 57 CAPLUS COPYRIGHT 2001 ACS

1998:258132 Document No. 129:49522 Adenosine A2A antagonist: a novel antiparkinsonian agent that does not provoke dyskinesia in **parkinsonian** monkeys. Kanda, Tomoyuki; Jackson, Michael J.; Smith, Lance A.; Pearce, Ronald K. B.; Nakamura, Joji; Kase, Hiroshi; Kuwana, Yoshihisa; Jenner, Peter (Neurodegenerative Disease Research Centre, Pharmacology Group, Biomedical Sciences Division, King's College London, London, SW3 6LX, UK). Ann. Neurol., 43(4), 507-513 (English)

1998. CODEN: ANNED3. ISSN: 0364-5134. Publisher: Lippincott-Raven Publishers.

AB Treatment of **Parkinson's** disease with L-DOPA therapy leads to long-term complications, including loss of drug efficacy and the onset of dyskinesia. Adenosine A2A receptors in the striatum are selectively localized to GABAergic output neurons of the striato-pallidal pathway and may avoid such problems. The novel adenosine A2A receptor antagonist KW-6002 was examd. for antiparkinsonian activity in MPTP-treated primates.

Oral administration of KW-6002 reversed motor disability in MPTP-treated common marmosets in a dose-dependent manner. However, KW-6002 only modestly increased overall locomotor activity and did not cause abnormal movement, such as stereotypy. The ability of KW-6002 to reverse motor disability was maintained on repeated daily administration for 21 days, and no tolerance was obsd. KW-6002 induced little or no dyskinesia in MPTP-treated primates previously primed to exhibit dyskinesia by prior exposure to L-DOPA. Thus, selective adenosine A2A receptor antagonists represent a new class of antiparkinsonian agents that do not produce hyperactivity or induce dyskinesia.

IT 155270-99-8

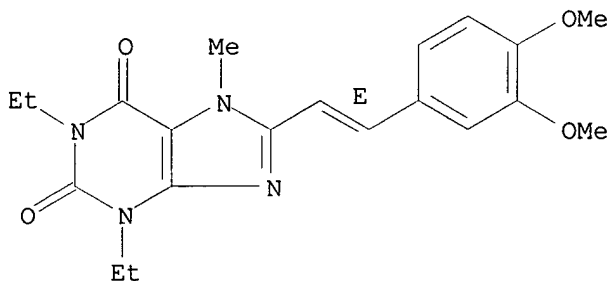
RL: BAC (Biological activity or effector, except adverse); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(adenosine A2A antagonist: novel antiparkinsonian agent that does not provoke dyskinesia in **parkinsonian** monkeys)

RN 155270-99-8 CAPLUS

CN 1H-Purine-2,6-dione, 8-[(1E)-2-(3,4-dimethoxyphenyl)ethenyl]-1,3-diethyl-3,7-dihydro-7-methyl- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



L22 ANSWER 26 OF 57 BIOSIS COPYRIGHT 2001 BIOSIS DUPLICATE 3
1999:120194 Document No.: PREV199900120194. Potential of adenosine A2A

receptor antagonists in the treatment of movement disorders. Mally, Judit;

Stone, Trevor W. (1). (1) West Medical Building, University Glasgow, Glasgow G12 8QQ UK. CNS Drugs, (Nov., 1998) Vol. 10, No. 5, pp. 311-320. ISSN: 1172-7047. Language: English.

L22 ANSWER 27 OF 57 CAPLUS COPYRIGHT 2001 ACS
1999:159011 Document No. 130:347294 A2A-selective adenosine receptor antagonists: development of water-soluble prodrugs and a new tritiated radioligand. Muller, Christa E.; Sauer, Roland; Maurinsh, Yuris; Huertas,

Rosa; Fulle, Friederike; Klotz, Karl-Norbert; Nagel, Jens; Hauber, Wolfgang (Institute of Pharmacy and Food Chemistry, University of Wurzburg, Wurzburg, Germany). Drug Dev. Res., 45(3/4), 190-197 (English) 1998. CODEN: DDREDK. ISSN: 0272-4391. Publisher: Wiley-Liss, Inc..

AB A2A adenosine receptor (AR) antagonists are promising new drugs for the treatment of **Parkinson's** disease. Further potential therapeutic indications for A2A AR antagonists include dementias, ischemias, and pain.

Potent, selective A2A AR antagonists have been developed, but their generally low water soly. is a major problem for conducting in vivo expts.

We developed a water-sol. phosphate prodrug (MSX-3) of a potent, selective

A2A AR antagonist (MSX-2), which is stable in aq. soln., but rapidly cleaved in vivo by phosphatases to release the active compd. MSX-2. Intracerebral application of MSX-3 led to a stimulation of motor activity in rats. Catalepsy, induced by pretreatment with either dopamine D1 or

D2 antagonists, was potently reversed by intracerebral application of MSX-3. A new A2A-selective antagonist radioligand, [³H]MSX-2, was prepd., which exhibits a KD value of 8 nM at rat brain striatal membranes.

IT 197075-91-5DP, derivs. 199680-78-9P 212131-38-9P

224622-14-4P 224622-16-6P 224622-25-7P

224622-27-9P 224622-30-4P 224622-32-6P

224622-36-0P 224622-43-9P

RL: BAC (Biological activity or effector, except adverse); BPR

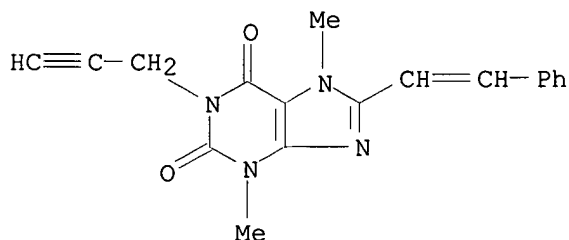
(Biological

process); PNU (Preparation, unclassified); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); PROC (Process); USES (Uses)

(water-sol. prodrugs of A2A-selective adenosine receptor antagonists and a new tritiated radioligand)

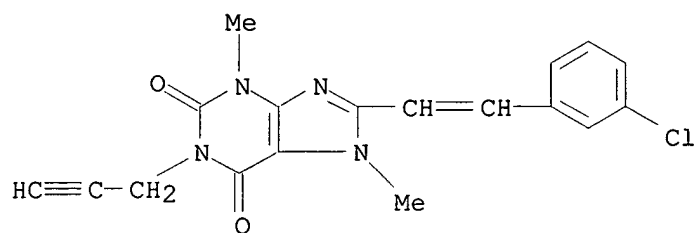
RN 197075-91-5 CAPLUS

CN 1H-Purine-2,6-dione, 3,7-dihydro-3,7-dimethyl-8-(2-phenylethenyl)-1-(2-propynyl)- (9CI) (CA INDEX NAME)

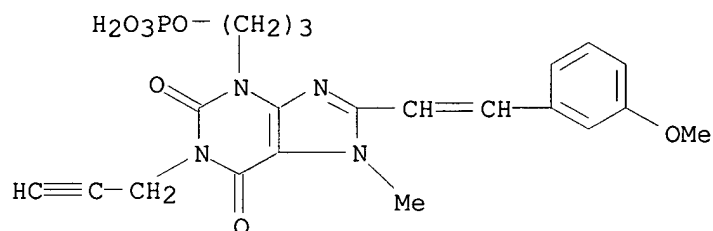


RN 199680-78-9 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(3-chlorophenyl)ethenyl]-3,7-dihydro-3,7-dimethyl-1-(2-propynyl)- (9CI) (CA INDEX NAME)

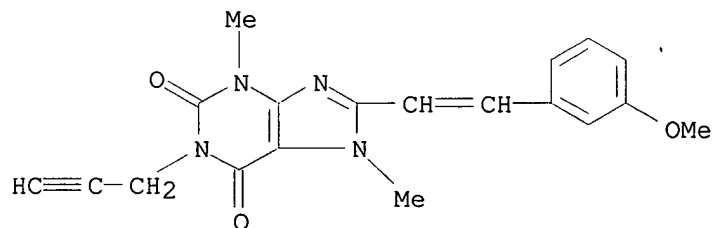


RN 212131-38-9 CAPLUS
 CN 1H-Purine-2,6-dione,
 3,7-dihydro-8-[2-(3-methoxyphenyl)ethenyl]-7-methyl-3-
 [3-(phosphonoxy)propyl]-1-(2-propynyl)-, disodium salt (9CI) (CA INDEX
 NAME)

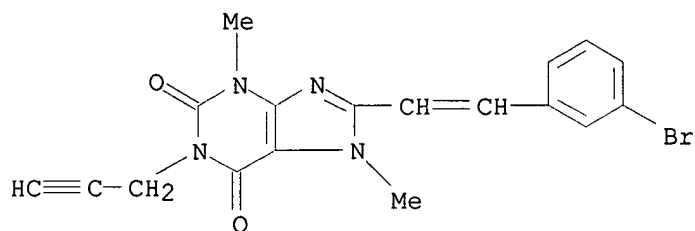


● 2 Na

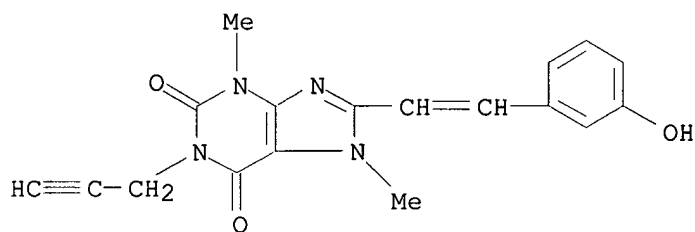
RN 224622-14-4 CAPLUS
 CN 1H-Purine-2,6-dione, 3,7-dihydro-8-[2-(3-methoxyphenyl)ethenyl]-3,7-
 dimethyl-1-(2-propynyl)- (9CI) (CA INDEX NAME)



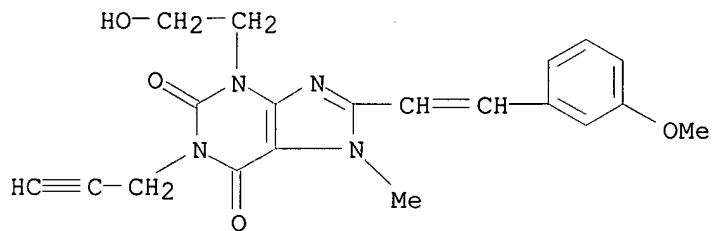
RN 224622-16-6 CAPLUS
 CN 1H-Purine-2,6-dione,
 8-[2-(3-bromophenyl)ethenyl]-3,7-dihydro-3,7-dimethyl-
 1-(2-propynyl)- (9CI) (CA INDEX NAME)



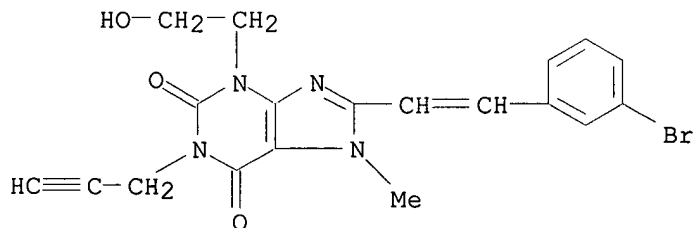
RN 224622-25-7 CAPLUS
 CN 1H-Purine-2,6-dione, 3,7-dihydro-8-[2-(3-hydroxyphenyl)ethenyl]-3,7-dimethyl-1-(2-propynyl)- (9CI) (CA INDEX NAME)



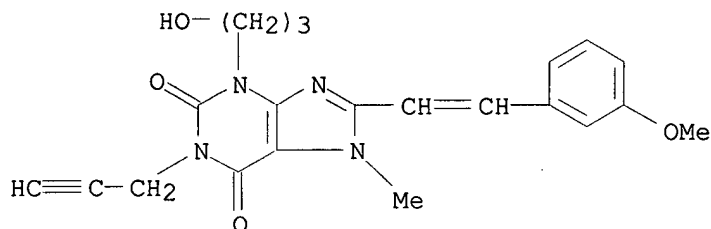
RN 224622-27-9 CAPLUS
 CN 1H-Purine-2,6-dione, 3,7-dihydro-3-(2-hydroxyethyl)-8-[2-(3-methoxyphenyl)ethenyl]-7-methyl-1-(2-propynyl)- (9CI) (CA INDEX NAME)



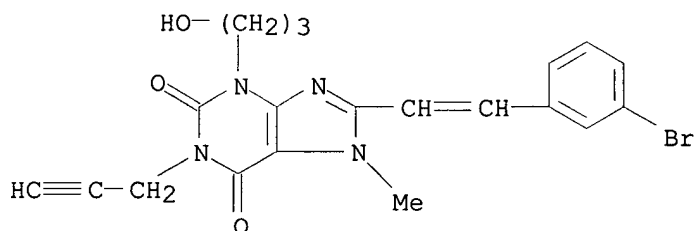
RN 224622-30-4 CAPLUS
 CN 1H-Purine-2,6-dione, 8-[2-(3-bromophenyl)ethenyl]-3,7-dihydro-3-(2-hydroxyethyl)-7-methyl-1-(2-propynyl)- (9CI) (CA INDEX NAME)



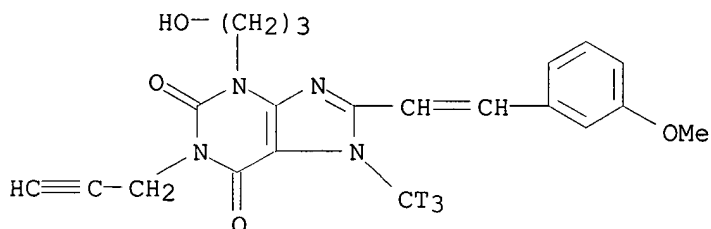
RN 224622-32-6 CAPLUS
 CN 1H-Purine-2,6-dione, 3,7-dihydro-3-(3-hydroxypropyl)-8-[2-(3-methoxyphenyl)ethenyl]-7-methyl-1-(2-propynyl)- (9CI) (CA INDEX NAME)



RN 224622-36-0 CAPLUS
 CN 1H-Purine-2,6-dione, 8-[2-(3-bromophenyl)ethenyl]-3,7-dihydro-3-(3-hydroxypropyl)-7-methyl-1-(2-propynyl)- (9CI) (CA INDEX NAME)



RN 224622-43-9 CAPLUS
 CN 1H-Purine-2,6-dione, 3,7-dihydro-3-(3-hydroxypropyl)-8-[2-(3-methoxyphenyl)ethenyl]-7-(methyl-t3)-1-(2-propynyl)- (9CI) (CA INDEX NAME)



L22 ANSWER 28 OF 57 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
 1999092885 EMBASE Sixth International Symposium on Adenosine and Adenine Nucleotides: New frontiers in the third millennium. Borea P.A.; Baraldi P.G.. P.A. Borea, University of Ferrara, Ferrara, Italy. Drug Development Research 45/3-4 (85) 1998.
 ISSN: 0272-4391. CODEN: DDREDK. Pub. Country: United States. Language: English.

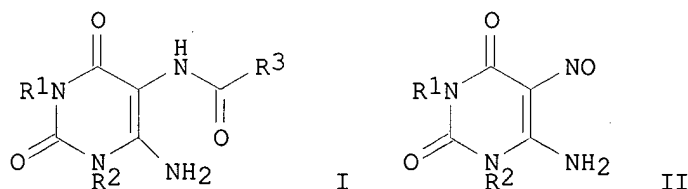
L22 ANSWER 29 OF 57 BIOSIS COPYRIGHT 2001 BIOSIS
1998:291795 Document No.: PREV199800291795. Water-soluble prodrugs of potent
A2A-selective adenosine receptor antagonists. Mueller, C. E. (1); Sauer,
R. (1); Maurinsh, Y. (1); Fuelle, F. (1); Nagel, J.; Hubuer, W.. (1)

Inst.

Pharmacy Food Chemistry, Univ. Wuerzburg Germany. Drug Development
Research, (Jan., 1998) Vol. 43, No. 1, pp. 33. Meeting Info.: 6th
International Symposium on Adenosine and Adenine Nucleotides: New
Frontiers in the 3rd Millennium Ferrara, Italy May 19-24, 1998 ISSN:
0272-4391. Language: English.

L22 ANSWER 30 OF 57 CAPLUS COPYRIGHT 2001 ACS
1997:187077 Document No. 126:212159 Preparation of uracil derivatives by
reduction and amidation. Miwa, Keiichi; Ito, Katsuhiko; Kato, Nobuyuki;
Kuge, Yukyasu; Kasai, Masaji; Tomioka, Shinji (Kyowa Hakko Kogyo Kk,
Japan). Jpn. Kokai Tokkyo Koho JP 09040652 A2 19970210 Heisei, 6 pp.
(Japanese). CODEN: JKXXAF. APPLICATION: JP 1995-192923 19950728.

GI



AB Claimed is a process for prepn. of the title compds. (I; R1, R2 = H,
lower alkyl; R3 = lower alkyl, cycloalkyl, etc.) by redn. of compds. (II; R1,
R2

= same as above) and then amidation with R3CO2H (R3 = same as above) or
their derivs. I are useful as intermediates in the prodn. of drugs for
treatment of dementia, urinary system diseases, and **Parkinson's**
diseases (no data). Thus, II (R1 = R2 = n-Pr) was treated with Na2S2O4
and then reacted with R3COCl [R3 = (E)-3,4-dimethoxycinnamyl] to give
69.4% I (R1, R2, R3 = same as above).

IT 141807-96-7P 155270-99-8P

RL: BAC (Biological activity or effector, except adverse); SPN (Synthetic
preparation); THU (Therapeutic use); BIOL (Biological study); PREP
(Preparation); USES (Uses)

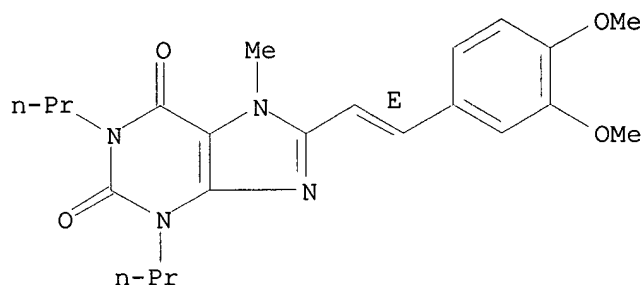
(prepn. of uracil derivs. by redn. and amidation)

RN 141807-96-7 CAPLUS

CN 1H-Purine-2,6-dione,

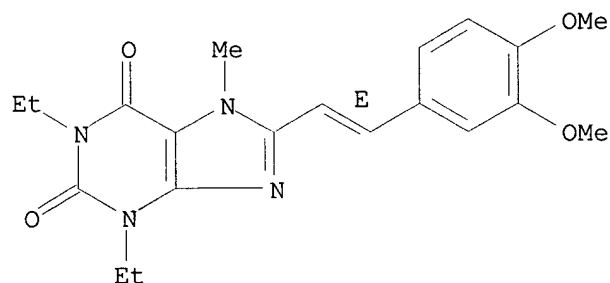
8-[(1E)-2-(3,4-dimethoxyphenyl)ethenyl]-3,7-dihydro-7-
methyl-1,3-dipropyl- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 155270-99-8 CAPLUS
 CN 1H-Purine-2,6-dione, 8-[(1E)-2-(3,4-dimethoxyphenyl)ethenyl]-1,3-diethyl-3,7-dihydro-7-methyl- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



L22 ANSWER 31 OF 57 BIOSIS COPYRIGHT 2001 BIOSIS
 1998:237416 Document No.: PREV199800237416. Functions of adenosine A2a receptors in striatum. Kuwana, Yoshihisa; Shiozaki, Shizuo; Nonaka, Hiromi; Kurokawa, Masako; Ichikawa, Shunji; Kanda, Tomoyuki; Koga, Kumiko; Aoyama, Shiro; Shiodou, Tomomi; Ochi, Mayumi; Ichimura, Michio; Mori, Akihisa; Richardson, P. J.; Kase, Hiroshi. Pharm. Res. Labs., Kyowa Hakko Kogyo, 1188 Shimotogari, Sunto-gun, Shizuoka 411 Japan. Okada, Y. [Editor]. International Congress Series, (1997) No. 1140, pp. 215. International Congress Series; The role of adenosine in the nervous system. Publisher: Elsevier Science Publishers B.V. PO Box 211, Sara Burgerhartstraat 25, 1000 AE Amsterdam, The Netherlands. Meeting Info.: International Symposium Kobe, Japan July 13-16, 1996 ISSN: 0531-5131. ISBN: 0-444-82643-2. Language: English.

L22 ANSWER 32 OF 57 BIOSIS COPYRIGHT 2001 BIOSIS
 1997:532232 Document No.: PREV199799831435. The role of adenosine A2A receptors in regulating GABAergic synaptic transmission in striatal medium spiny neurons. Mori, A.; Shindou, T.; Ochi, M.; Ichimura, M.; Nonaka, H.; Kase, H.. Pharm. Res. Labs, Kyowa Hakko Kogyo Co. Ltd., Shizuoka 411 Japan. Society for Neuroscience Abstracts, (1997) Vol. 23, No. 1-2, pp. 1962. Meeting Info.: 27th Annual Meeting of the Society for Neuroscience

New Orleans, Louisiana, USA October 25-30, 1997 ISSN: 0190-5295.
Language:
English.

L22 ANSWER 33 OF 57 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
97197515 EMBASE Document No.: 1997197515. **Alzheimer's** disease and related Dementias: Prospects for treatment. Williams M.; Davis R.E.. M. Williams, NUDRD 464, Abbott Laboratories, 100 Abbott Park Road, Abbott Park, IL, 60064-3500, United States. mike.williams@abbott.com. Expert Opinion on Investigational Drugs 6/6 (735-757) 1997.
Refs: 62.
ISSN: 1354-3784. CODEN: EOIDER. Pub. Country: United Kingdom. Language: English. Summary Language: English.

AB **Alzheimer's** disease (AD) represents a major challenge to healthcare costs and to academic and pharmaceutical research efforts. The approval in 1996 of the first of the second generation acetylcholinesterase inhibitors, donepezil (Aricept(TM); Eisai/Pfizer), has offered new hope, albeit palliative, to AD sufferers and care givers. Research has continued on the genetics of AD with the identification of the autosomal dominant inheritance of genetic defects in one of three distinct genes coding for the presenilins 1 and 2 and amyloid precursor protein (APP). While driving an ever increasing research effort related to the production, deposition and clearance of A.beta. peptides, these mutations account for less than 10% of the AD cases reported, indicating that other causative factors, both genetic and environmental, may contribute to the pathophysiology of AD unrelated to familial cohorts. A newly developed transgenic mouse model and a broader appreciation of the multifactorial nature of this complex, chronic disease state may help provide a more objective approach to understanding the disease per se as opposed to amyloid neurotoxicity specifically which may or may not be causative.

L22 ANSWER 34 OF 57 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
97290673 EMBASE Document No.: 1997290673. Adenosine A(2A) receptor antagonists as new agents for the treatment of **Parkinson's** disease. Richardson P.J.; Kase H.; Jenner P.G.. P.J. Richardson, Department of Pharmacology, Tennis Court Road, Cambridge CB2 1QJ, United Kingdom. Trends in Pharmacological Sciences 18/9 (338-344) 1997.
Refs: 48.
ISSN: 0165-6147. CODEN: TPHSDY.
Publisher Ident.: S 0165-6147(97)01096-1. Pub. Country: United Kingdom. Language: English. Summary Language: English.

AB There is now good reason to believe that blockade of the adenosine A, receptor could be of value in the treatment of **Parkinson's** disease. Peter J. Richardson, Hiroshi Kase and Peter G. Jenner review the actions of this receptor in the striatum, emphasizing its ability to modulate the neuronal activity of striatal GABA-releasing output neurones, and showing that recently developed A(2A) receptor antagonists are capable of reducing the disabling effects of nigral cell degeneration in primates. They conclude that such antagonists may be useful as novel therapeutic agents for the treatment of **Parkinson's** disease.

L22 ANSWER 35 OF 57 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
97290666 EMBASE Document No.: 1997290666. Improved therapies for
Parkinson's disease: Life beyond dopamine D2/D3 receptor agonists
(multiple letters) [1]. Williams M.; Wright S.; Lloyd G.K.. M. Williams,
Pharmaceutical Products Division, Abbott Laboratories, Abbott Park, IL
60065-3500, United States. Trends in Pharmacological Sciences 18/9
(308-310) 1997.
Refs: 43.
ISSN: 0165-6147. CODEN: TPHSDY. Pub. Country: United Kingdom. Language:
English.

L22 ANSWER 36 OF 57 BIOSIS COPYRIGHT 2001 BIOSIS
1997:468281 Document No.: PREV199799767484. A2A adenosine receptor
antagonists
are antiparkinsonian in animal models. Kuwana, Y.; Shiozaki, S.; Kanda,
T.; Kurokawa, M.; Koga, K.; Ochi, M.; Ikeda, K.; Jenner, P.. Pharm. Res.
Lab., Kyowa Hakko Kogyo, 1188 Shimotogari, Suntogun, Shizuoka 411 Japan.
Society for Neuroscience Abstracts, (1997) Vol. 23, No. 1-2, pp. 297.
Meeting Info.: 27th Annual Meeting of the Society for Neuroscience, Part
1
New Orleans, Louisiana, USA October 25-30, 1997 ISSN: 0190-5295.
Language:
English.

L22 ANSWER 37 OF 57 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
97364156 EMBASE Document No.: 1997364156. Adenosine A(2A) receptors and
neuroprotection. Ongini E.; Adami M.; Ferri C.; Bertorelli R.. E. Ongini,
Schering-Plough Research Institute, San Raffaele Science Park, Via
Olgettina 58, I-20132 Milan, Italy. Annals of the New York Academy of
Sciences 825/- (30-48) 1997.
Refs: 86.
ISSN: 0077-8923. CODEN: ANYAA. Pub. Country: United States. Language:
English. Summary Language: English.

AB The adenosine A(2A) receptor subtype is one of the four adenosine
receptors that have been identified in the mammalian organism. In
addition
to being found in blood vessels, platelets and polymorphonuclear
leukocytes, the A(2A) receptors are abundant in the central nervous
system, especially in the striatum. The recent development of selective
A(2A) receptor ligands, in particular of receptor antagonists, makes it
possible to elucidate the function of A(2A) receptors in normal and
altered conditions. Pharmacological studies have shown that A(2A)
receptor
antagonists are potentially effective for treatment of neurodegenerative
processes such as **Parkinson's** disease. Their activity is
attributed to the close anatomical and functional links between A(2A)
receptors and dopaminergic pathways in the basal ganglia. More recently,
A(2A) receptor antagonists have proved to be active in models of cerebral
ischemia. While the mechanisms underlying the role of A(2A) receptors in
the hypoxia/ischemia processes remains to be clarified, it is recognized
that A(2A) receptor antagonists counteract the effects of excitatory
aminoacids, which are massively released after cerebral ischemia. Another
function of A(2A) receptors is related to protection from seizures, but
further studies are needed to elucidate their specific interaction, if

any, with neuronal excitability. Altogether, the great advance recently made with the discovery of selective A(2A) receptor ligands provides increasing information on the function of A(2A) receptors and opens new perspectives for treatment of neurological disorders.

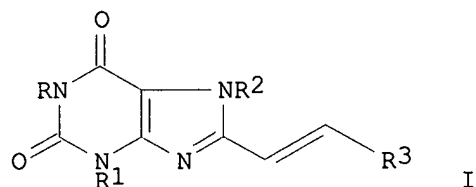
L22 ANSWER 38 OF 57 CAPLUS COPYRIGHT 2001 ACS

1996:113590 Document No. 124:289111 Preparation of xanthine derivatives for treatment of **Parkinson's** disease. Suzuki, Fumio; Shimada, Junichi; Koike, Nobuaki; Nakamura, Joji; Shioazaki, Shizuo; Ichikawa, Shunji; Ishii, Akio; Nonaka, Hiromi (Kyowa Hakko Kogyo Co., Ltd., Japan). U.S. US 5484920 A 19960116, 61 pp. Cont.-in-part of U.S. Ser. No.

42,535,

abandoned. (English). CODEN: USXXAM. APPLICATION: US 1993-133510 19931007. PRIORITY: JP 1992-257834 19920928; US 1993-42535 19930405; JP 1993-236176 19930922.

GI



AB Xanthines I [R, R1 = Me, Et; R2 = H, alkyl; R3 = substituted Ph] were prep'd. as selective adenosine A2 antagonists. Thus, 5,6-diamino-1,3-diethyluracil was treated with 3,4-dimethoxycinnamic acid to give I [R = R1 = Et, R2 = H, R3 = 3,4-(MeO)2C6H3] which at 1X10⁻⁵ M caused 98% inhibition of A2 receptor activity.

IT 141807-96-7P 141807-97-8P 142665-35-8P
147700-16-1P 147700-32-1P 147700-51-4P
151539-22-9P 151539-46-7P 151539-54-7P
151539-58-1P 151539-61-6P 155270-98-7P
155270-99-8P 155271-04-8P 155271-06-0P
155271-07-1P 155271-10-6P 155271-12-8P
155271-18-4P 155271-20-8P 155271-24-2P
155271-26-4P 155271-28-6P 155271-30-0P
155271-32-2P 155271-34-4P 155271-36-6P
155271-38-8P 155271-40-2P 155271-42-4P
155271-44-6P 155271-46-8P 155271-48-0P
155271-50-4P 155271-52-6P 155271-54-8P
155271-56-0P 155271-58-2P 155271-60-6P
155271-62-8P 155271-64-0P 155271-66-2P
155271-68-4P 155271-70-8P 155271-72-0P
155271-74-2P 155271-76-4P 155271-78-6P
155271-80-0P 155272-03-0P 155272-05-2P
155814-31-6P 155814-33-8P 169958-98-9P

RL: BAC (Biological activity or effector, except adverse); RCT (Reactant);

SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)

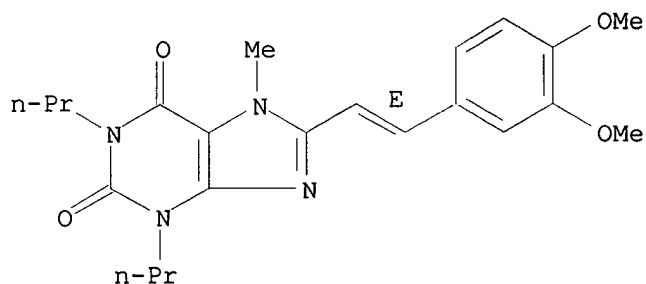
(prepn. of arylvinylxanthines as selective A2 receptor antagonists)

RN 141807-96-7 CAPLUS

CN 1H-Purine-2,6-dione,

8-[(1E)-2-(3,4-dimethoxyphenyl)ethenyl]-3,7-dihydro-7-methyl-1,3-dipropyl- (9CI) (CA INDEX NAME)

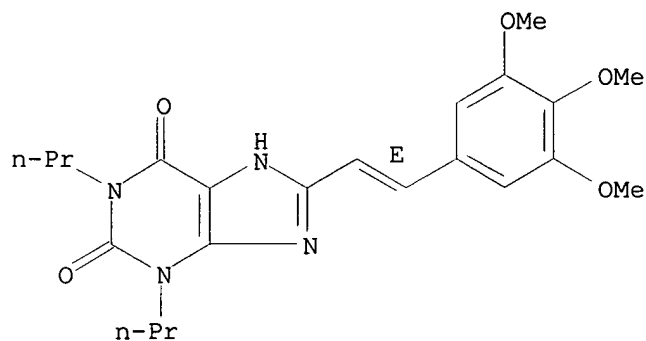
Double bond geometry as shown.



RN 141807-97-8 CAPLUS

CN 1H-Purine-2,6-dione, 3,7-dihydro-1,3-dipropyl-8-[2-(3,4,5-trimethoxyphenyl)ethenyl]-, (E)- (9CI) (CA INDEX NAME)

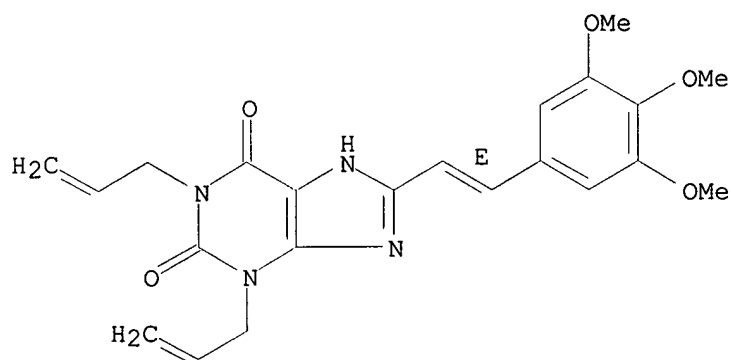
Double bond geometry as shown.



RN 142665-35-8 CAPLUS

CN 1H-Purine-2,6-dione, 3,7-dihydro-1,3-di-2-propenyl-8-[2-(3,4,5-trimethoxyphenyl)ethenyl]-, (E)- (9CI) (CA INDEX NAME)

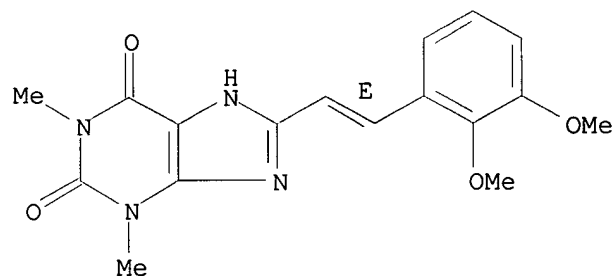
Double bond geometry as shown.



RN 147700-16-1 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(2,3-dimethoxyphenyl)ethenyl]-3,7-dihydro-1,3-dimethyl-, (E)- (9CI) (CA INDEX NAME)

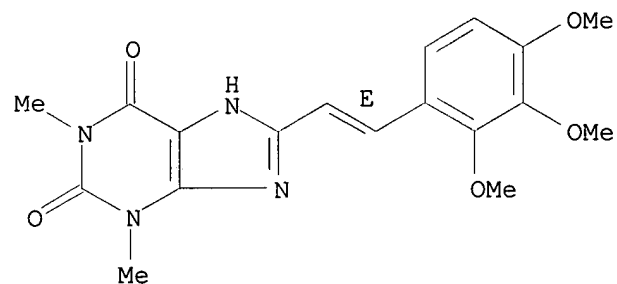
Double bond geometry as shown.



RN 147700-32-1 CAPLUS

CN 1H-Purine-2,6-dione, 3,7-dihydro-1,3-dimethyl-8-[2-(2,3,4-trimethoxyphenyl)ethenyl]-, (E)- (9CI) (CA INDEX NAME)

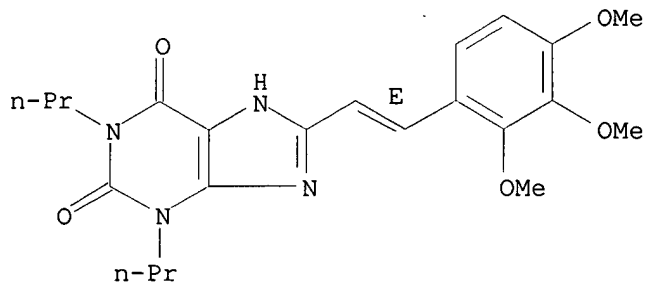
Double bond geometry as shown.



RN 147700-51-4 CAPLUS

CN 1H-Purine-2,6-dione, 3,7-dihydro-1,3-dipropyl-8-[2-(2,3,4-trimethoxyphenyl)ethenyl]-, (E)- (9CI) (CA INDEX NAME)

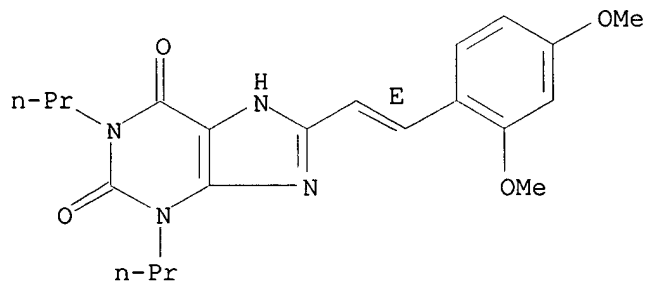
Double bond geometry as shown.



RN 151539-22-9 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(2,4-dimethoxyphenyl)ethenyl]-3,7-dihydro-1,3-dipropyl-, (E)- (9CI) (CA INDEX NAME)

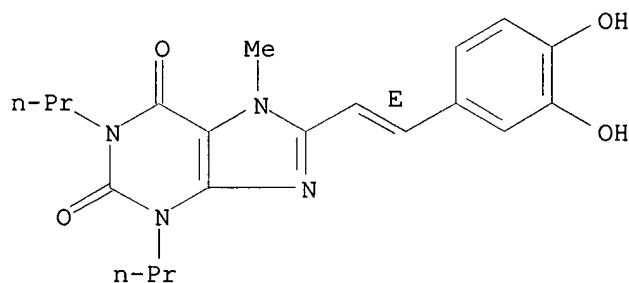
Double bond geometry as shown.



RN 151539-46-7 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(3,4-dihydroxyphenyl)ethenyl]-3,7-dihydro-7-methyl-1,3-dipropyl-, (E)- (9CI) (CA INDEX NAME)

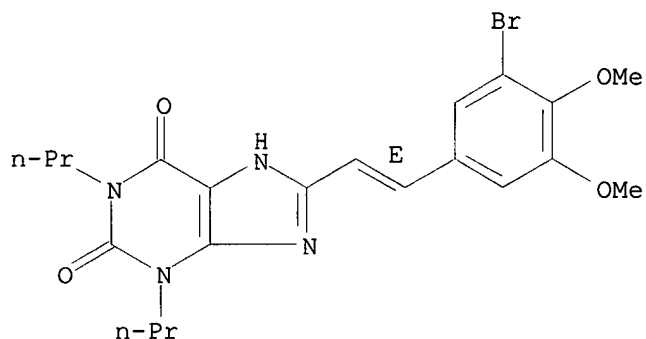
Double bond geometry as shown.



RN 151539-54-7 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(3-bromo-4,5-dimethoxyphenyl)ethenyl]-3,7-dihydro-1,3-dipropyl-, (E)- (9CI) (CA INDEX NAME)

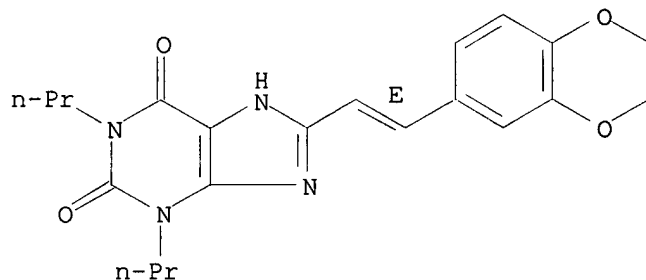
Double bond geometry as shown.



RN 151539-58-1 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(2,3-dihydro-1,4-benzodioxin-6-yl)ethenyl]-3,7-dihydro-1,3-dipropyl-, (E)- (9CI) (CA INDEX NAME)

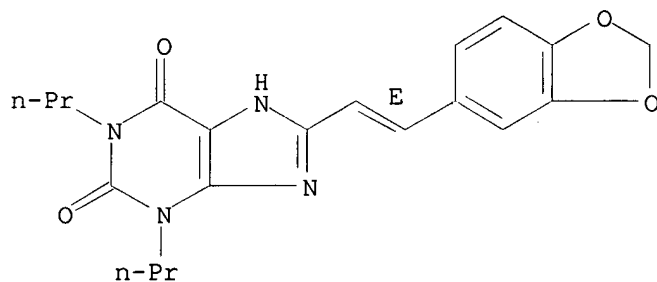
Double bond geometry as shown.



RN 151539-61-6 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(1,3-benzodioxol-5-yl)ethenyl]-3,7-dihydro-1,3-dipropyl-, (E)- (9CI) (CA INDEX NAME)

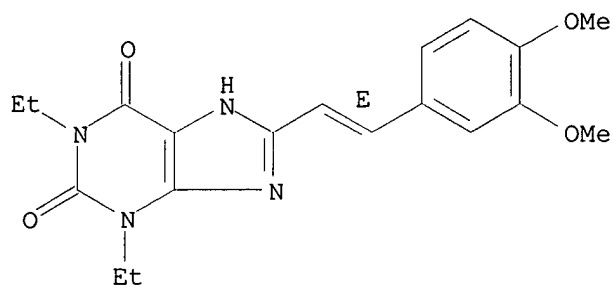
Double bond geometry as shown.



RN 155270-98-7 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(3,4-dimethoxyphenyl)ethenyl]-1,3-diethyl-3,7-dihydro-, (E)- (9CI) (CA INDEX NAME)

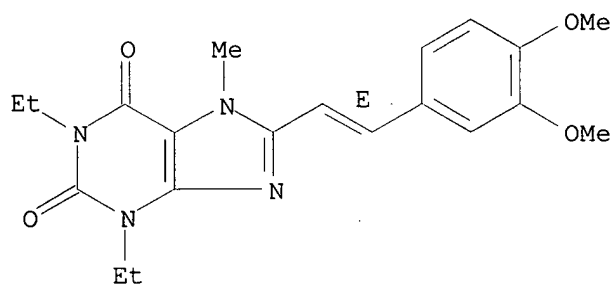
Double bond geometry as shown.



RN 155270-99-8 CAPLUS

CN 1H-Purine-2,6-dione, 8-[(1E)-2-(3,4-dimethoxyphenyl)ethenyl]-1,3-diethyl-3,7-dihydro-7-methyl- (9CI) (CA INDEX NAME)

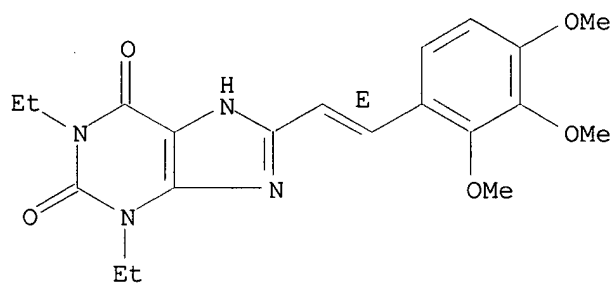
Double bond geometry as shown.



RN 155271-04-8 CAPLUS

CN 1H-Purine-2,6-dione, 1,3-diethyl-3,7-dihydro-8-[2-(2,3,4-trimethoxyphenyl)ethenyl]-, (E)- (9CI) (CA INDEX NAME)

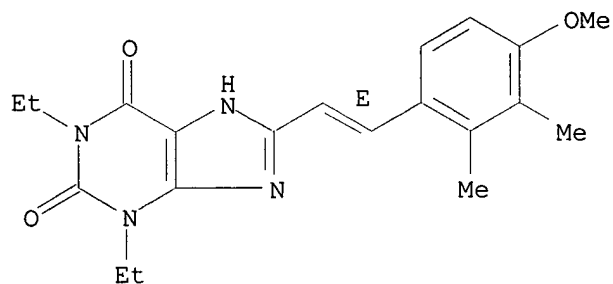
Double bond geometry as shown.



RN 155271-06-0 CAPLUS

CN 1H-Purine-2,6-dione, 1,3-diethyl-3,7-dihydro-8-[2-(4-methoxy-2,3-dimethylphenyl)ethenyl]-, (E)- (9CI) (CA INDEX NAME)

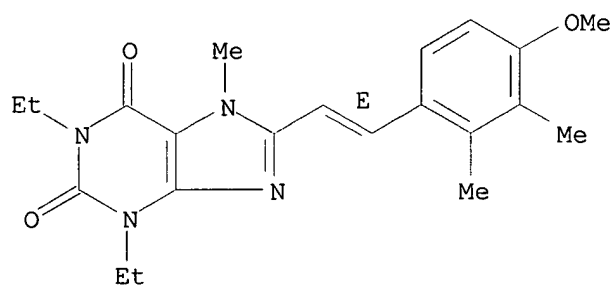
Double bond geometry as shown.



RN 155271-07-1 CAPLUS

CN 1H-Purine-2,6-dione, 1,3-diethyl-3,7-dihydro-8-[2-(4-methoxy-2,3-dimethylphenyl)ethenyl]-7-methyl-, (E)- (9CI) (CA INDEX NAME)

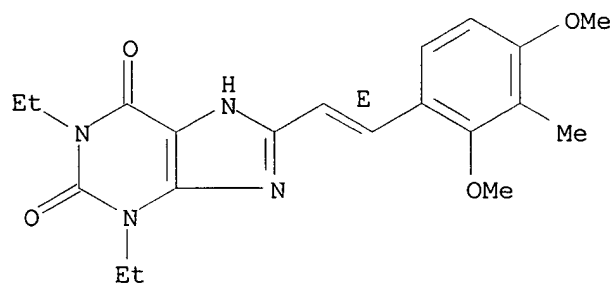
Double bond geometry as shown.



RN 155271-10-6 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(2,4-dimethoxy-3-methylphenyl)ethenyl]-1,3-diethyl-3,7-dihydro-, (E)- (9CI) (CA INDEX NAME)

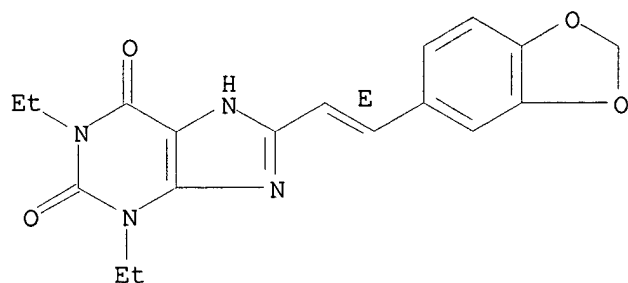
Double bond geometry as shown.



RN 155271-12-8 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(1,3-benzodioxol-5-yl)ethenyl]-1,3-diethyl-3,7-dihydro-, (E)- (9CI) (CA INDEX NAME)

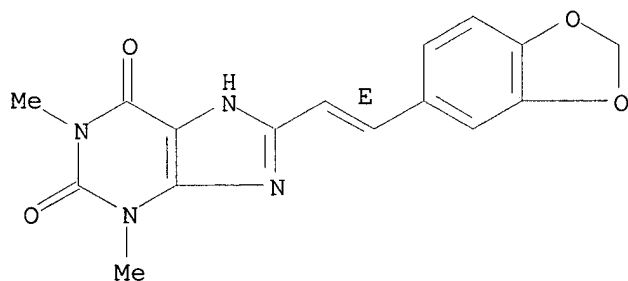
Double bond geometry as shown.



RN 155271-18-4 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(1,3-benzodioxol-5-yl)ethenyl]-3,7-dihydro-1,3-dimethyl-, (E)- (9CI) (CA INDEX NAME)

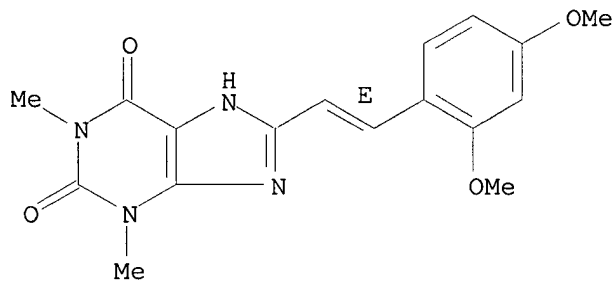
Double bond geometry as shown.



RN 155271-20-8 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(2,4-dimethoxyphenyl)ethenyl]-3,7-dihydro-1,3-dimethyl-, (E)- (9CI) (CA INDEX NAME)

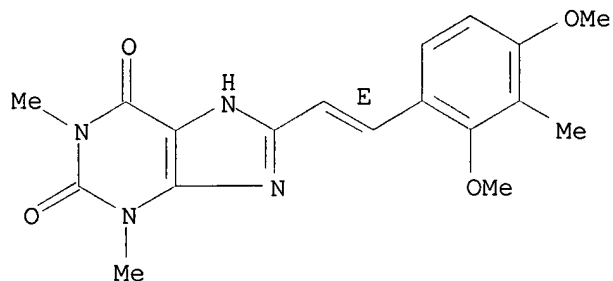
Double bond geometry as shown.



RN 155271-24-2 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(2,4-dimethoxy-3-methylphenyl)ethenyl]-3,7-dihydro-1,3-dimethyl-, (E)- (9CI) (CA INDEX NAME)

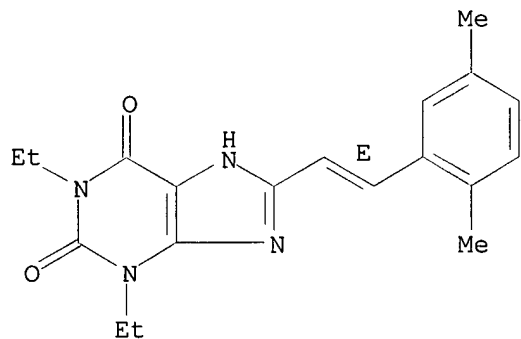
Double bond geometry as shown.



RN 155271-26-4 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(2,5-dimethylphenyl)ethenyl]-1,3-diethyl-3,7-dihydro-, (E)- (9CI) (CA INDEX NAME)

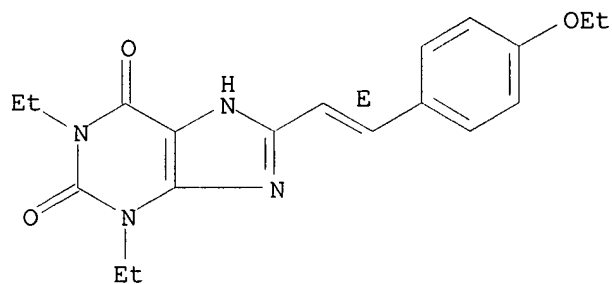
Double bond geometry as shown.



RN 155271-28-6 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(4-ethoxyphenyl)ethenyl]-1,3-diethyl-3,7-dihydro-, (E)- (9CI) (CA INDEX NAME)

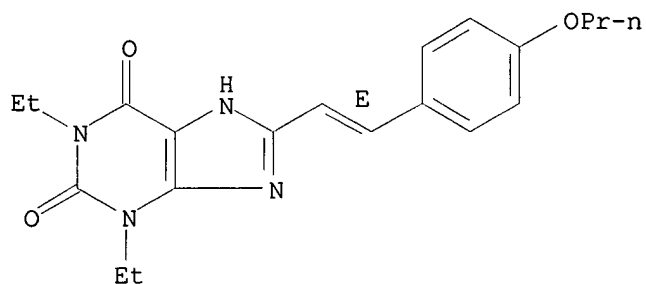
Double bond geometry as shown.



RN 155271-30-0 CAPLUS

CN 1H-Purine-2,6-dione, 1,3-diethyl-3,7-dihydro-8-[2-(4-propoxyphenyl)ethenyl]-, (E)- (9CI) (CA INDEX NAME)

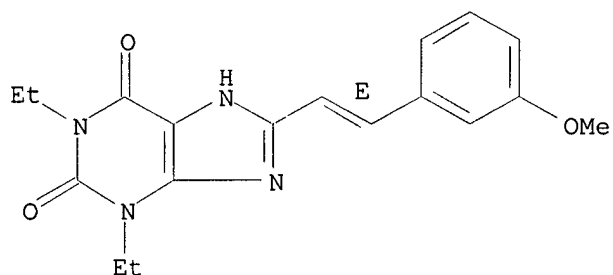
Double bond geometry as shown.



RN 155271-32-2 CAPLUS

CN 1H-Purine-2,6-dione, 1,3-diethyl-3,7-dihydro-8-[2-(3-methoxyphenyl)ethenyl]-, (E)- (9CI) (CA INDEX NAME)

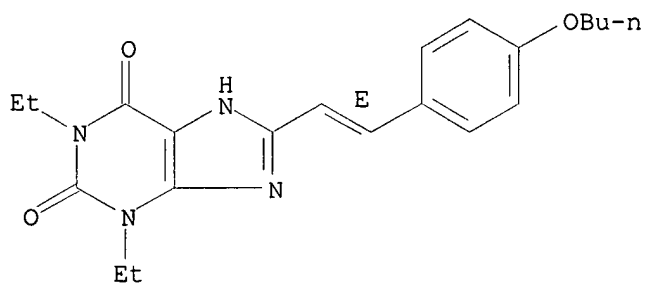
Double bond geometry as shown.



RN 155271-34-4 CAPLUS

CN 1H-Purine-2,6-dione,
8-[2-(4-butoxyphenyl)ethenyl]-1,3-diethyl-3,7-dihydro-
, (E)- (9CI) (CA INDEX NAME)

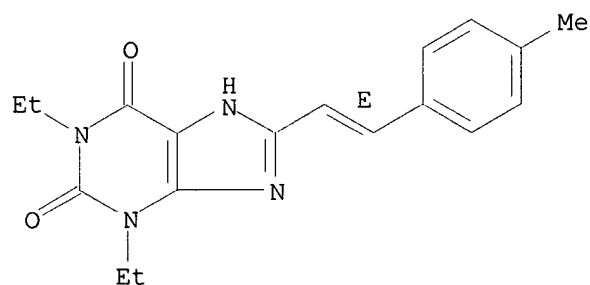
Double bond geometry as shown.



RN 155271-36-6 CAPLUS

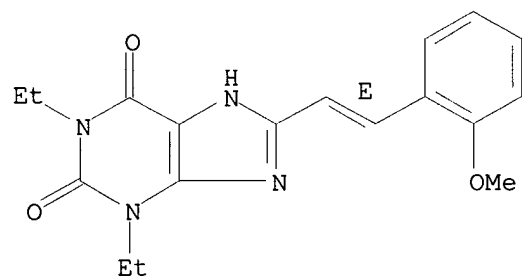
CN 1H-Purine-2,6-dione,
1,3-diethyl-3,7-dihydro-8-[2-(4-methylphenyl)ethenyl]-
, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



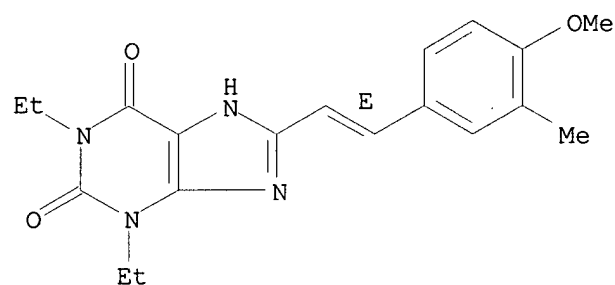
RN 155271-38-8 CAPLUS
CN 1H-Purine-2,6-dione, 1,3-diethyl-3,7-dihydro-8-[2-(2-methoxyphenyl)ethenyl]-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



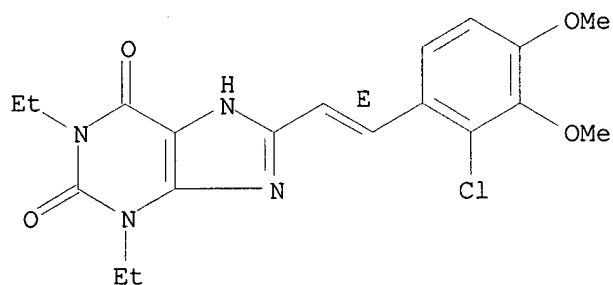
RN 155271-40-2 CAPLUS
CN 1H-Purine-2,6-dione, 1,3-diethyl-3,7-dihydro-8-[2-(4-methoxy-3-methylphenyl)ethenyl]-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 155271-42-4 CAPLUS
CN 1H-Purine-2,6-dione, 8-[2-(2-chloro-3,4-dimethoxyphenyl)ethenyl]-1,3-diethyl-3,7-dihydro-, (E)- (9CI) (CA INDEX NAME)

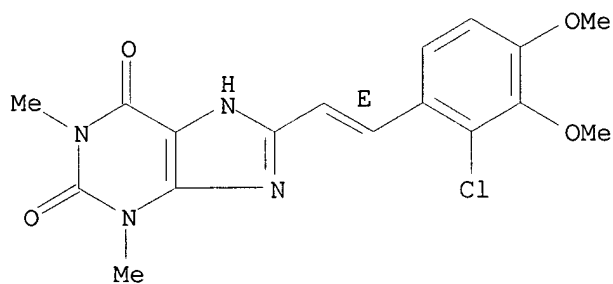
Double bond geometry as shown.



RN 155271-44-6 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(2-chloro-3,4-dimethoxyphenyl)ethenyl]-3,7-dihydro-1,3-dimethyl-, (E)- (9CI) (CA INDEX NAME)

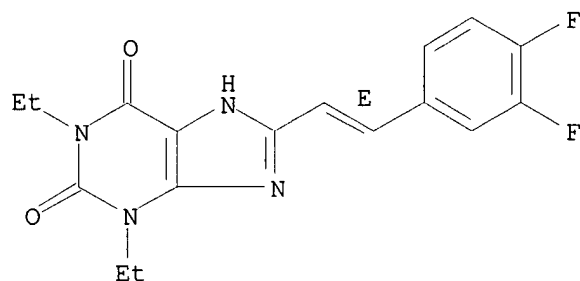
Double bond geometry as shown.



RN 155271-46-8 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(3,4-difluorophenyl)ethenyl]-1,3-diethyl-3,7-dihydro-, (E)- (9CI) (CA INDEX NAME)

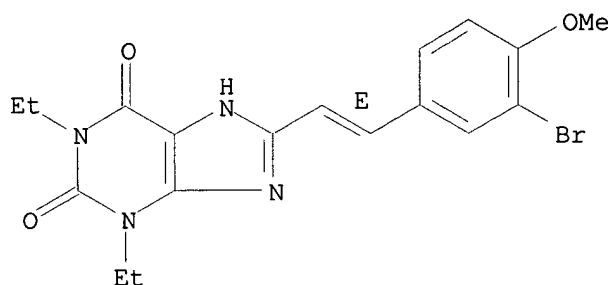
Double bond geometry as shown.



RN 155271-48-0 CAPLUS

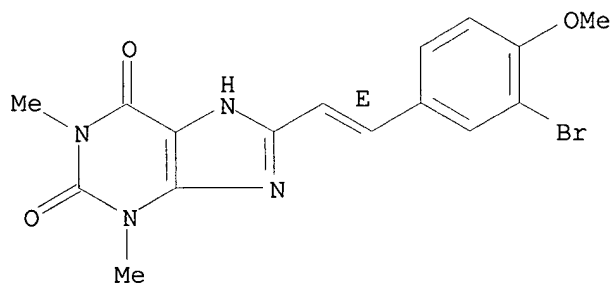
CN 1H-Purine-2,6-dione, 8-[2-(3-bromo-4-methoxyphenyl)ethenyl]-1,3-diethyl-3,7-dihydro-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



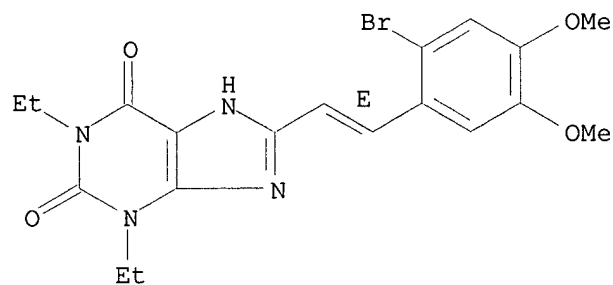
RN 155271-50-4 CAPLUS
 CN 1H-Purine-2,6-dione, 8-[2-(3-bromo-4-methoxyphenyl)ethenyl]-3,7-dihydro-1,3-dimethyl-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



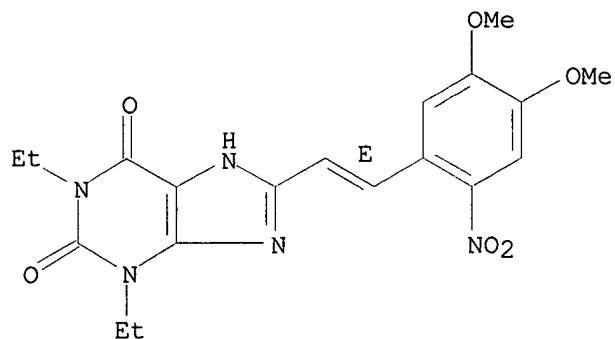
RN 155271-52-6 CAPLUS
 CN 1H-Purine-2,6-dione, 8-[2-(2-bromo-4,5-dimethoxyphenyl)ethenyl]-1,3-diethyl-3,7-dihydro-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



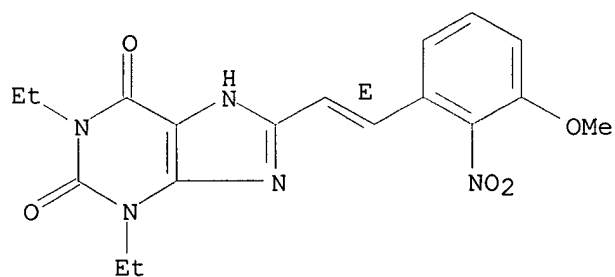
RN 155271-54-8 CAPLUS
 CN 1H-Purine-2,6-dione, 8-[2-(4,5-dimethoxy-2-nitrophenyl)ethenyl]-1,3-diethyl-3,7-dihydro-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



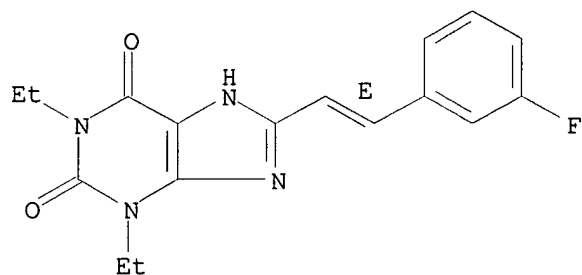
RN 155271-56-0 CAPLUS
 CN 1H-Purine-2,6-dione, 1,3-diethyl-3,7-dihydro-8-[2-(3-methoxy-2-nitrophenyl)ethenyl]-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



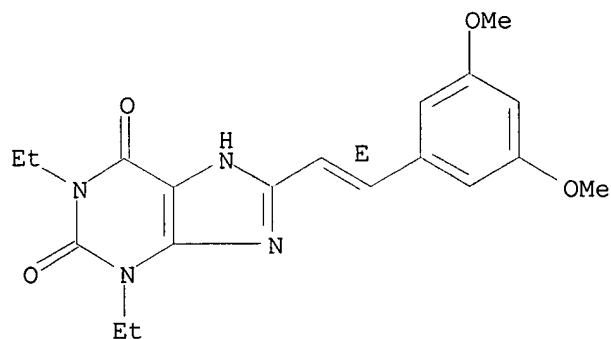
RN 155271-58-2 CAPLUS
 CN 1H-Purine-2,6-dione, 1,3-diethyl-8-[2-(3-fluorophenyl)ethenyl]-3,7-dihydro-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



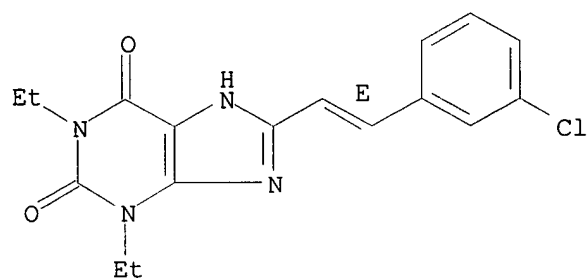
RN 155271-60-6 CAPLUS
 CN 1H-Purine-2,6-dione, 8-[2-(3,5-dimethoxyphenyl)ethenyl]-1,3-diethyl-3,7-dihydro-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



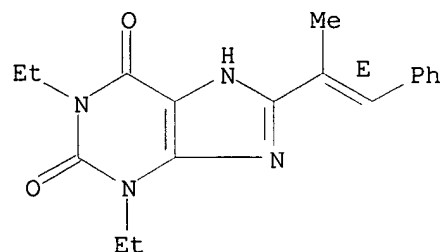
RN 155271-62-8 CAPLUS
CN 1H-Purine-2,6-dione,
8-[2-(3-chlorophenyl)ethenyl]-1,3-diethyl-3,7-dihydro-
, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 155271-64-0 CAPLUS
CN 1H-Purine-2,6-dione,
1,3-diethyl-3,7-dihydro-8-(1-methyl-2-phenylethenyl)-
, (E)- (9CI) (CA INDEX NAME)

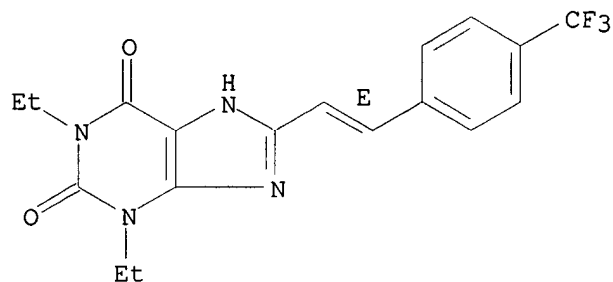
Double bond geometry as shown.



RN 155271-66-2 CAPLUS
CN 1H-Purine-2,6-dione, 1,3-diethyl-3,7-dihydro-8-[2-[4-

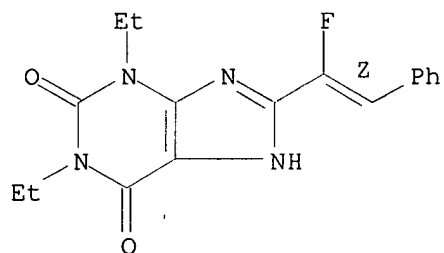
(trifluoromethyl)phenyl]ethenyl]-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



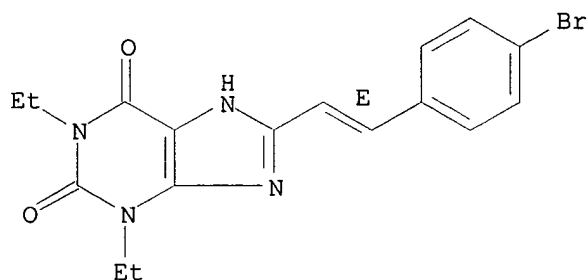
RN 155271-68-4 CAPLUS
CN 1H-Purine-2,6-dione,
1,3-diethyl-8-(1-fluoro-2-phenylethenyl)-3,7-dihydro-
, (Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 155271-70-8 CAPLUS
CN 1H-Purine-2,6-dione,
8-[2-(4-bromophenyl)ethenyl]-1,3-diethyl-3,7-dihydro-
, (E)- (9CI) (CA INDEX NAME)

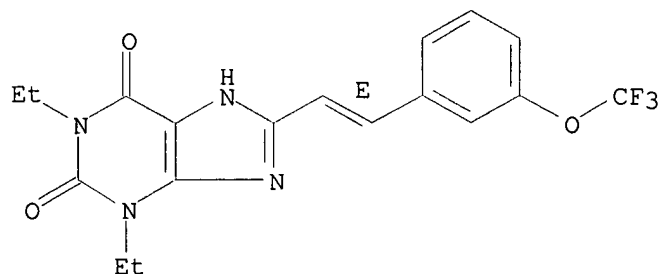
Double bond geometry as shown.



RN 155271-72-0 CAPLUS
CN 1H-Purine-2,6-dione, 1,3-diethyl-3,7-dihydro-8-[2-[3-

(trifluoromethoxy)phenyl]ethenyl]-, (E)- (9CI) (CA INDEX NAME)

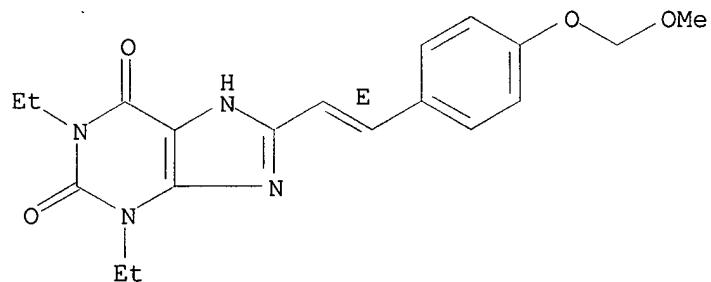
Double bond geometry as shown.



RN 155271-74-2 CAPLUS

CN 1H-Purine-2,6-dione, 1,3-diethyl-3,7-dihydro-8-[2-[4-(methoxymethoxy)phenyl]ethenyl]-, (E)- (9CI) (CA INDEX NAME)

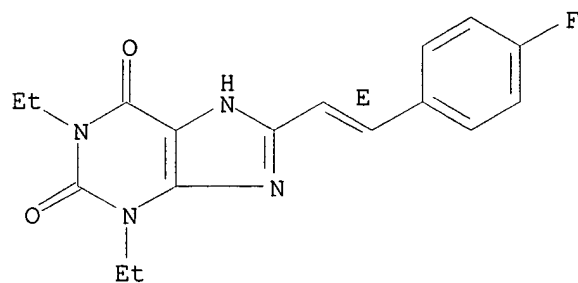
Double bond geometry as shown.



RN 155271-76-4 CAPLUS

CN 1H-Purine-2,6-dione, 1,3-diethyl-8-[2-(4-fluorophenyl)ethenyl]-3,7-dihydro-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

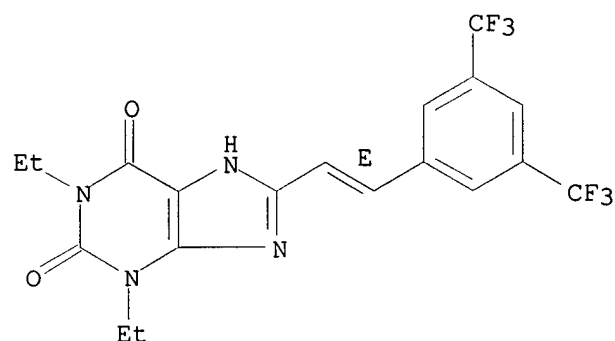


RN 155271-78-6 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-[3,5-bis(trifluoromethyl)phenyl]ethenyl]-1,3-

diethyl-3,7-dihydro-, (E)- (9CI) (CA INDEX NAME)

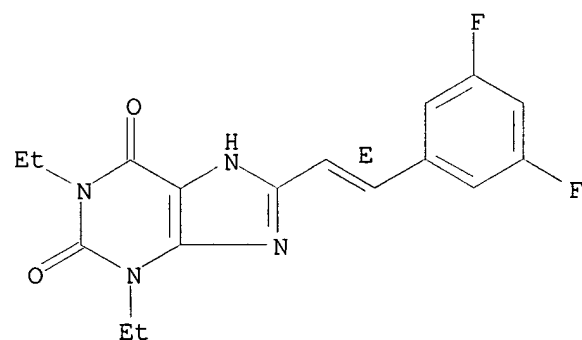
Double bond geometry as shown.



RN 155271-80-0 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(3,5-difluorophenyl)ethenyl]-1,3-diethyl-3,7-dihydro-, (E)- (9CI) (CA INDEX NAME)

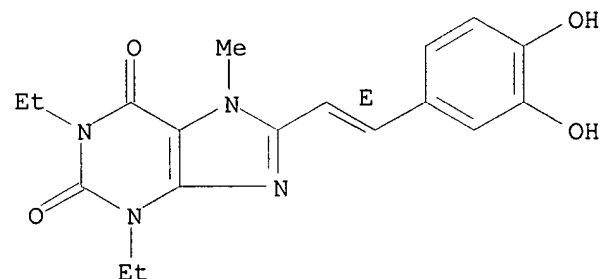
Double bond geometry as shown.



RN 155272-03-0 CAPLUS

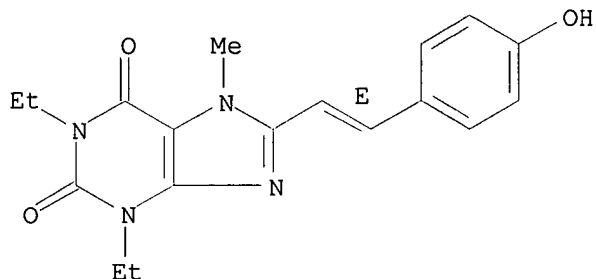
CN 1H-Purine-2,6-dione, 8-[2-(3,4-dihydroxyphenyl)ethenyl]-1,3-diethyl-3,7-dihydro-7-methyl-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



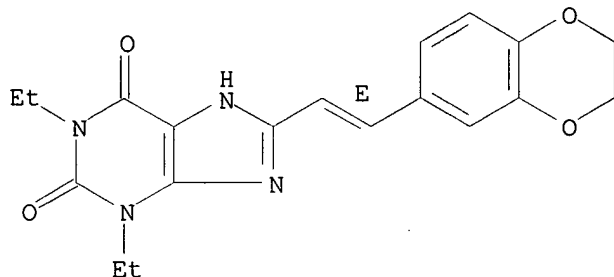
RN 155272-05-2 CAPLUS
CN 1H-Purine-2,6-dione, 1,3-diethyl-3,7-dihydro-8-[2-(4-hydroxyphenyl)ethenyl]-7-methyl-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



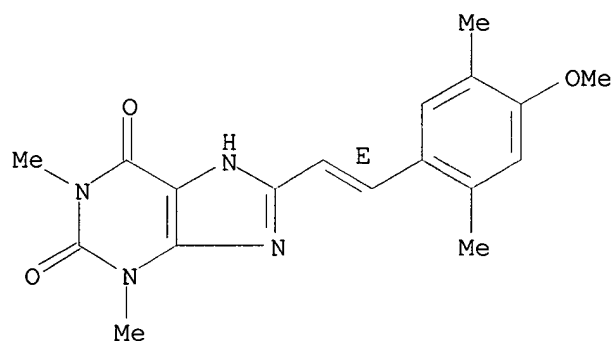
RN 155814-31-6 CAPLUS
CN 1H-Purine-2,6-dione, 8-[2-(2,3-dihydro-1,4-benzodioxin-6-yl)ethenyl]-1,3-diethyl-3,7-dihydro-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 155814-33-8 CAPLUS
CN 1H-Purine-2,6-dione, 3,7-dihydro-8-[2-(4-methoxy-2,5-dimethylphenyl)ethenyl]-1,3-dimethyl-, (E)- (9CI) (CA INDEX NAME)

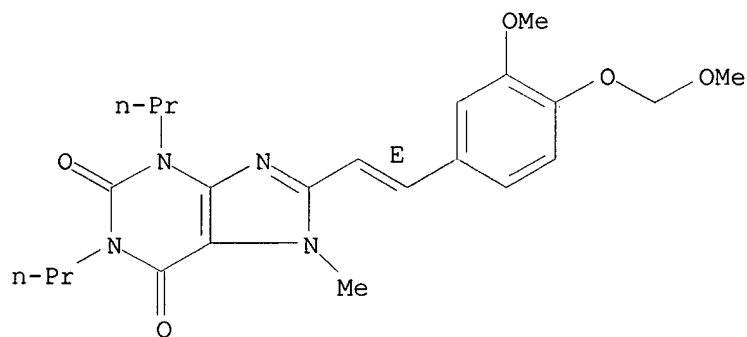
Double bond geometry as shown.



RN 169958-98-9 CAPLUS

CN 1H-Purine-2,6-dione, 3,7-dihydro-8-[2-[3-methoxy-4-(methoxymethoxy)phenyl]ethenyl]-7-methyl-1,3-dipropyl-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



IT 51389-37-8P 141807-86-5P 141807-94-5P
 141807-98-9P 142665-36-9P 142665-38-1P
 147700-17-2P 147700-33-2P 147700-34-3P
 147700-40-1P 147700-52-5P 147700-54-7P
 151539-19-4P 151539-21-8P 151539-23-0P
 151539-30-9P 151539-31-0P 151539-42-3P
 151539-44-5P 151539-60-5P 151539-62-7P
 155271-01-5P 155271-03-7P 155271-05-9P
 155271-09-3P 155271-11-7P 155271-13-9P
 155271-16-2P 155271-17-3P 155271-19-5P
 155271-21-9P 155271-25-3P 155271-27-5P
 155271-29-7P 155271-31-1P 155271-33-3P
 155271-35-5P 155271-37-7P 155271-39-9P
 155271-41-3P 155271-43-5P 155271-45-7P
 155271-47-9P 155271-49-1P 155271-51-5P
 155271-53-7P 155271-55-9P 155271-57-1P
 155271-59-3P 155271-61-7P 155271-63-9P
 155271-65-1P 155271-67-3P 155271-69-5P
 155271-71-9P 155271-73-1P 155271-75-3P

155271-77-5P 155271-79-7P 155271-83-3P
 155271-85-5P 155271-96-8P 155272-00-7P
 155272-02-9P 155272-06-3P 155814-25-8P
 155814-26-9P 155814-28-1P 155814-32-7P
 155814-34-9P 175675-60-2P

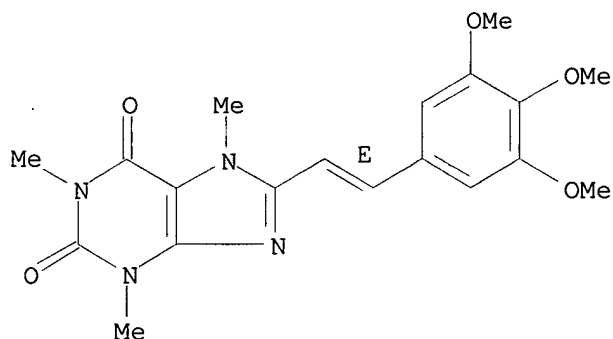
RL: BAC (Biological activity or effector, except adverse); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)

(prepn. of arylvinylxanthines as selective A2 receptor antagonists)

RN 51389-37-8 CAPLUS

CN 1H-Purine-2,6-dione, 3,7-dihydro-1,3,7-trimethyl-8-[(1E)-2-(3,4,5-trimethoxyphenyl)ethenyl]- (9CI) (CA INDEX NAME)

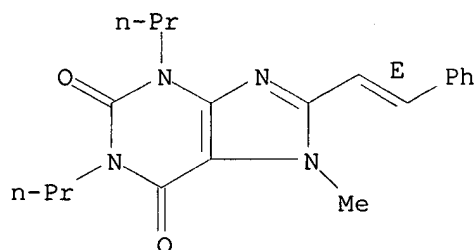
Double bond geometry as shown.



RN 141807-86-5 CAPLUS

CN 1H-Purine-2,6-dione, 3,7-dihydro-7-methyl-8-(2-phenylethenyl)-1,3-dipropyl-, (E)- (9CI) (CA INDEX NAME)

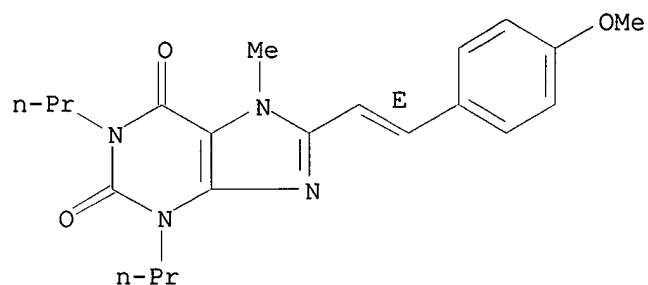
Double bond geometry as shown.



RN 141807-94-5 CAPLUS

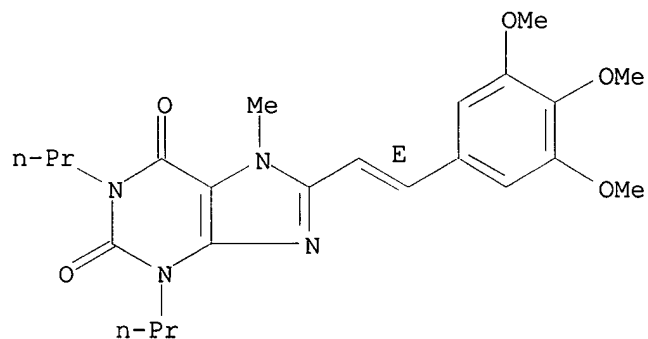
CN 1H-Purine-2,6-dione, 3,7-dihydro-8-[2-(4-methoxyphenyl)ethenyl]-7-methyl-1,3-dipropyl-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



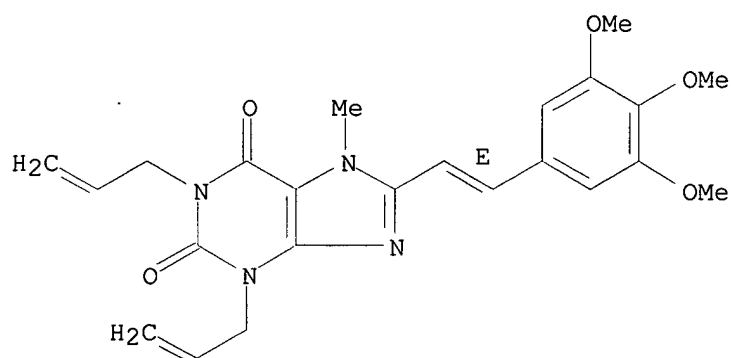
RN 141807-98-9 CAPLUS
 CN 1H-Purine-2,6-dione, 3,7-dihydro-7-methyl-1,3-dipropyl-8-[2-(3,4,5-trimethoxyphenyl)ethenyl]-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 142665-36-9 CAPLUS
 CN 1H-Purine-2,6-dione, 3,7-dihydro-7-methyl-1,3-di-2-propenyl-8-[(1E)-2-(3,4,5-trimethoxyphenyl)ethenyl]- (9CI) (CA INDEX NAME)

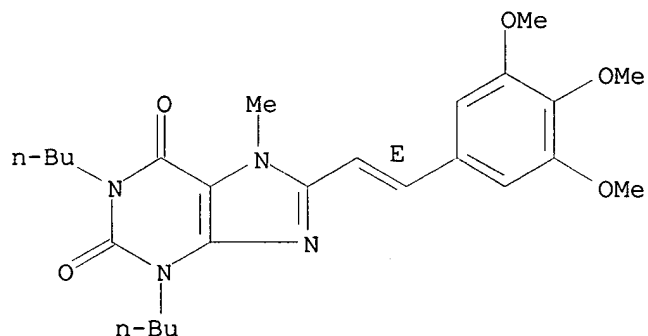
Double bond geometry as shown.



RN 142665-38-1 CAPLUS

CN 1H-Purine-2,6-dione, 1,3-dibutyl-3,7-dihydro-7-methyl-8-[2-(3,4,5-trimethoxyphenyl)ethenyl]-, (E)- (9CI) (CA INDEX NAME)

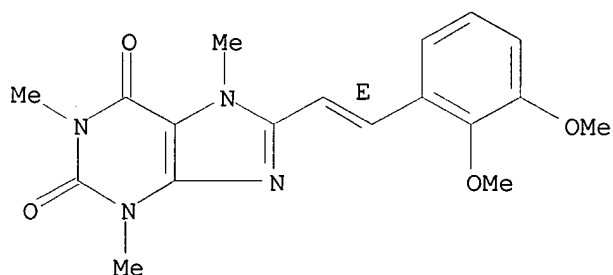
Double bond geometry as shown.



RN 147700-17-2 CAPLUS

CN 1H-Purine-2,6-dione,
8-[2-(2,3-dimethoxyphenyl)ethenyl]-3,7-dihydro-1,3,7-trimethyl-, (E)- (9CI) (CA INDEX NAME)

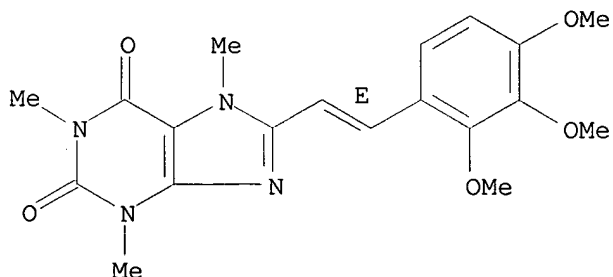
Double bond geometry as shown.



RN 147700-33-2 CAPLUS

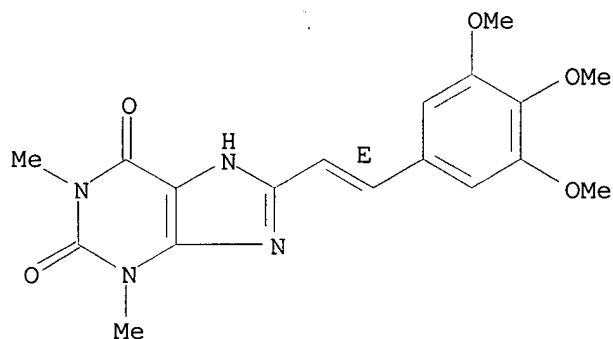
CN 1H-Purine-2,6-dione, 3,7-dihydro-1,3,7-trimethyl-8-[(1E)-2-(2,3,4-trimethoxyphenyl)ethenyl]- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



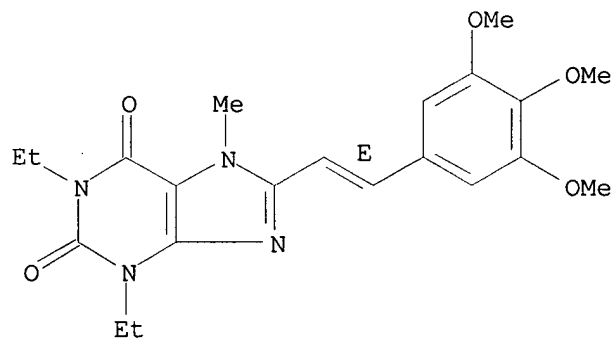
RN 147700-34-3 CAPLUS
CN 1H-Purine-2,6-dione, 3,7-dihydro-1,3-dimethyl-8-[(1E)-2-(3,4,5-trimethoxyphenyl)ethenyl]- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



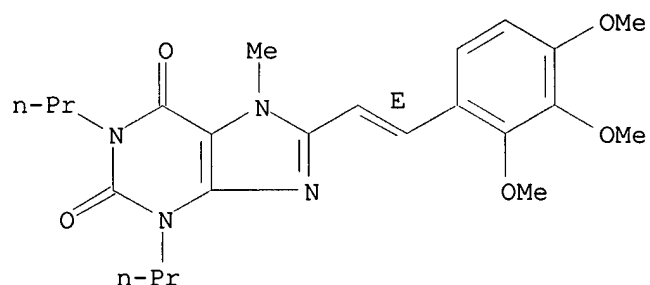
RN 147700-40-1 CAPLUS
CN 1H-Purine-2,6-dione, 1,3-diethyl-3,7-dihydro-7-methyl-8-[(1E)-2-(3,4,5-trimethoxyphenyl)ethenyl]- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



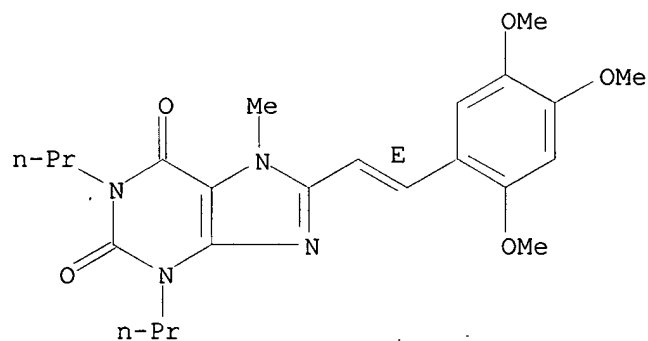
RN 147700-52-5 CAPLUS
CN 1H-Purine-2,6-dione, 3,7-dihydro-7-methyl-1,3-dipropyl-8-[2-(2,3,4-trimethoxyphenyl)ethenyl]-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



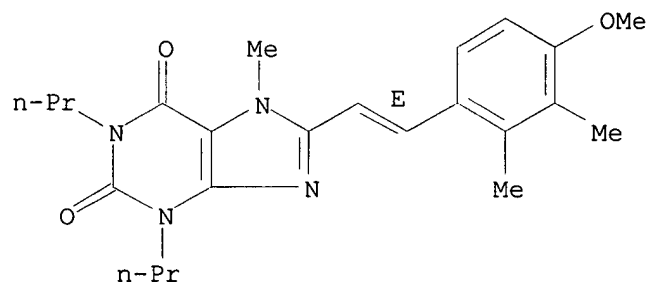
RN 147700-54-7 CAPLUS
 CN 1H-Purine-2,6-dione, 3,7-dihydro-7-methyl-1,3-dipropyl-8-[2-(2,4,5-trimethoxyphenyl)ethenyl]-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



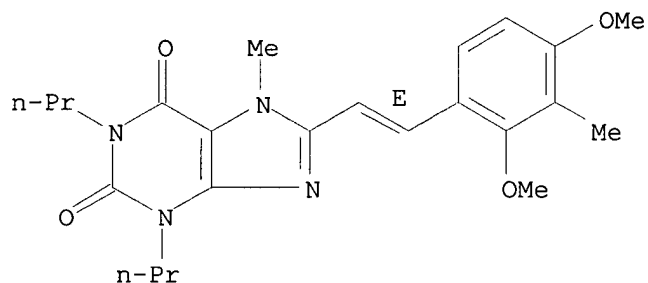
RN 151539-19-4 CAPLUS
 CN 1H-Purine-2,6-dione, 3,7-dihydro-8-[2-(4-methoxy-2,3-dimethylphenyl)ethenyl]-7-methyl-1,3-dipropyl-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 151539-21-8 CAPLUS
 CN 1H-Purine-2,6-dione, 8-[2-(2,4-dimethoxy-3-methylphenyl)ethenyl]-3,7-dihydro-7-methyl-1,3-dipropyl-, (E)- (9CI) (CA INDEX NAME)

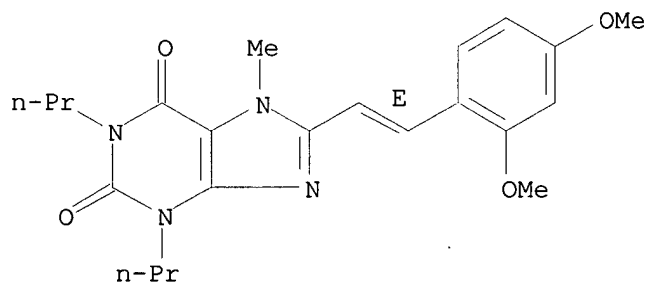
Double bond geometry as shown.



RN 151539-23-0 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(2,4-dimethoxyphenyl)ethenyl]-3,7-dihydro-7-methyl-1,3-dipropyl-, (E)- (9CI) (CA INDEX NAME)

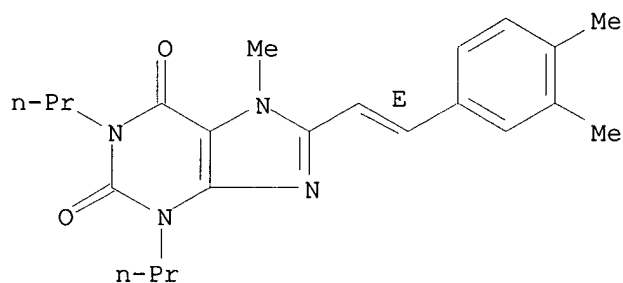
Double bond geometry as shown.



RN 151539-30-9 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(3,4-dimethylphenyl)ethenyl]-3,7-dihydro-7-methyl-1,3-dipropyl-, (E)- (9CI) (CA INDEX NAME)

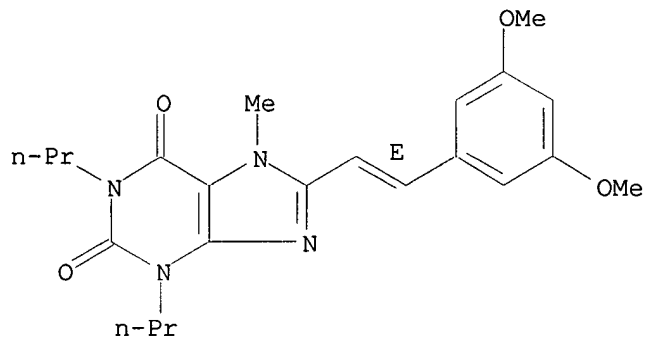
Double bond geometry as shown.



RN 151539-31-0 CAPLUS

CN 1H-Purine-2,6-dione, 8-[(1E)-2-(3,5-dimethoxyphenyl)ethenyl]-3,7-dihydro-7-methyl-1,3-dipropyl-, (9CI) (CA INDEX NAME)

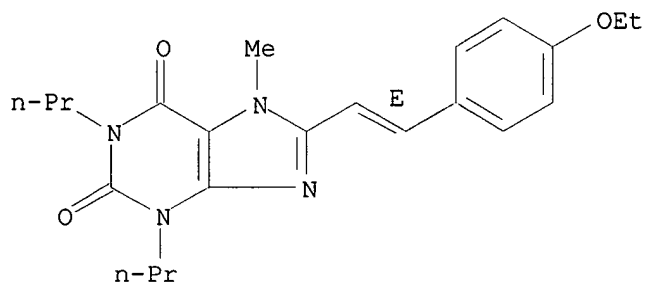
Double bond geometry as shown.



RN 151539-42-3 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(4-ethoxyphenyl)ethenyl]-3,7-dihydro-7-methyl-1,3-dipropyl-, (E)- (9CI) (CA INDEX NAME)

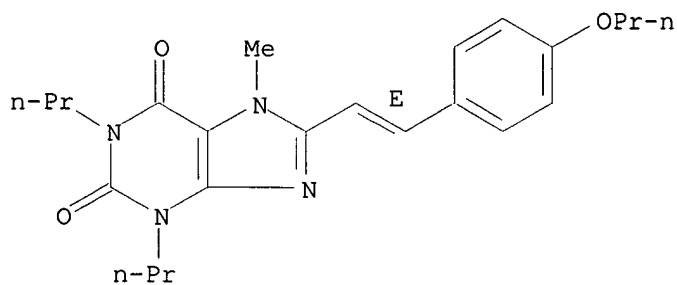
Double bond geometry as shown.



RN 151539-44-5 CAPLUS

CN 1H-Purine-2,6-dione, 3,7-dihydro-7-methyl-8-[2-(4-propoxyphenyl)ethenyl]-1,3-dipropyl-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

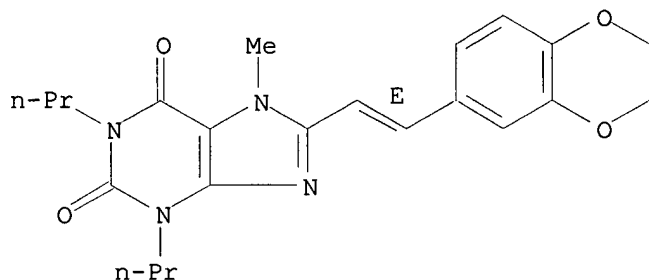


RN 151539-60-5 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(2,3-dihydro-1,4-benzodioxin-6-yl)ethenyl]-3,7-

dihydro-7-methyl-1,3-dipropyl-, (E)- (9CI) (CA INDEX NAME)

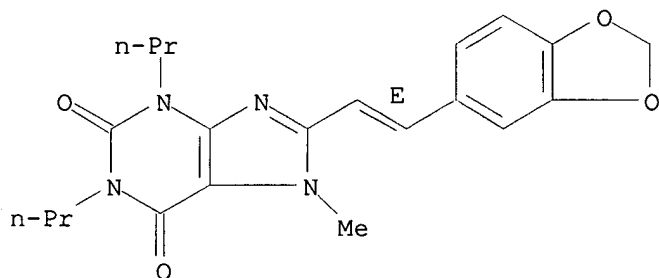
Double bond geometry as shown.



RN 151539-62-7 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(1,3-benzodioxol-5-yl)ethenyl]-3,7-dihydro-7-methyl-1,3-dipropyl-, (E)- (9CI) (CA INDEX NAME)

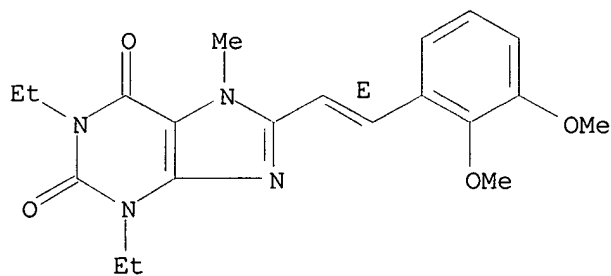
Double bond geometry as shown.



RN 155271-01-5 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(2,3-dimethoxyphenyl)ethenyl]-1,3-diethyl-3,7-dihydro-7-methyl-, (E)- (9CI) (CA INDEX NAME)

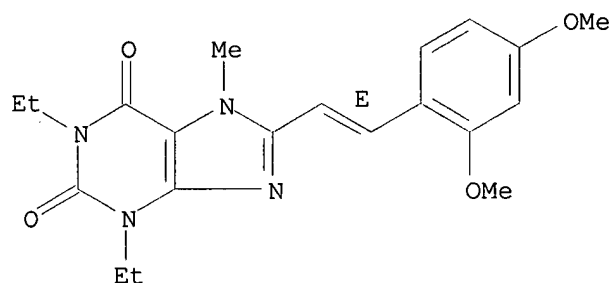
Double bond geometry as shown.



RN 155271-03-7 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(2,4-dimethoxyphenyl)ethenyl]-1,3-diethyl-3,7-dihydro-7-methyl-, (E)- (9CI) (CA INDEX NAME)

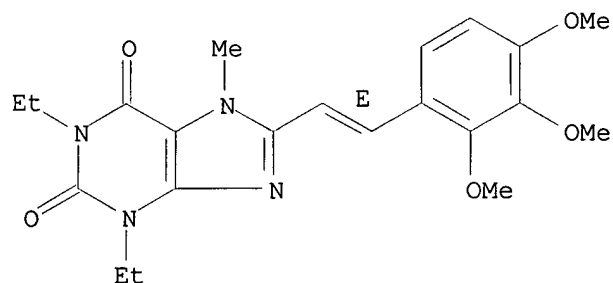
Double bond geometry as shown.



RN 155271-05-9 CAPLUS

CN 1H-Purine-2,6-dione, 1,3-diethyl-3,7-dihydro-7-methyl-8-[2-(2,3,4-trimethoxyphenyl)ethenyl]-, (E)- (9CI) (CA INDEX NAME)

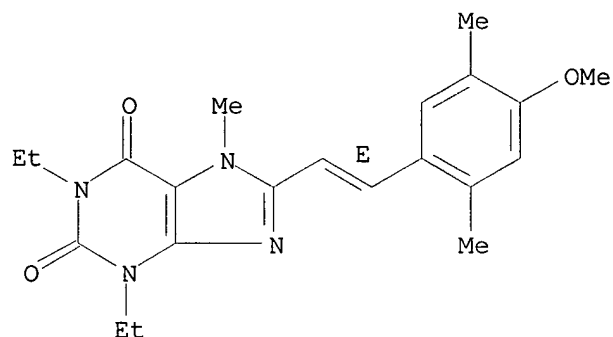
Double bond geometry as shown.



RN 155271-09-3 CAPLUS

CN 1H-Purine-2,6-dione, 1,3-diethyl-3,7-dihydro-8-[2-(4-methoxy-2,5-dimethylphenyl)ethenyl]-7-methyl-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

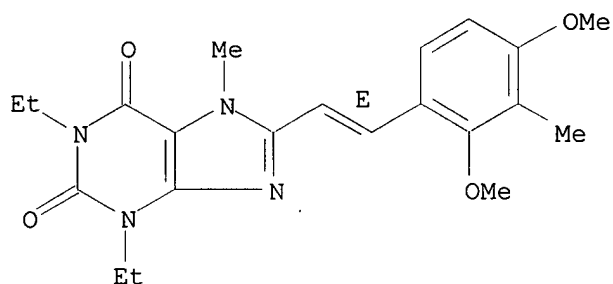


RN 155271-11-7 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(2,4-dimethoxy-3-methylphenyl)ethenyl]-1,3-

diethyl-3,7-dihydro-7-methyl-, (E)- (9CI) (CA INDEX NAME)

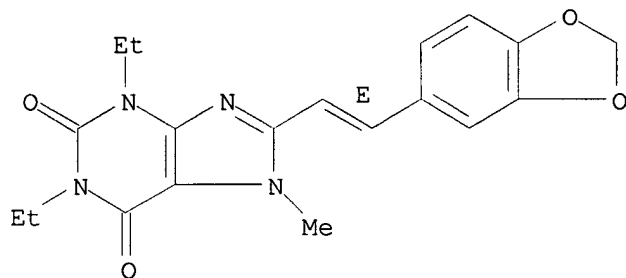
Double bond geometry as shown.



RN 155271-13-9 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(1,3-benzodioxol-5-yl)ethenyl]-1,3-diethyl-3,7-dihydro-7-methyl-, (E)- (9CI) (CA INDEX NAME)

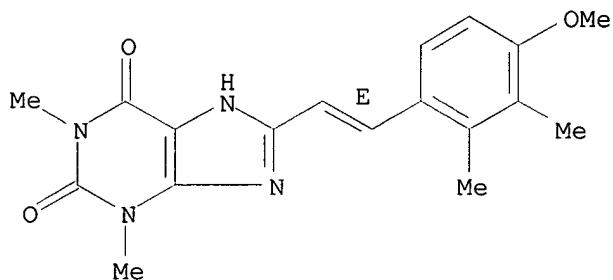
Double bond geometry as shown.



RN 155271-16-2 CAPLUS

CN 1H-Purine-2,6-dione, 3,7-dihydro-8-[(1E)-2-(4-methoxy-2,3-dimethylphenyl)ethenyl]-1,3-dimethyl- (9CI) (CA INDEX NAME)

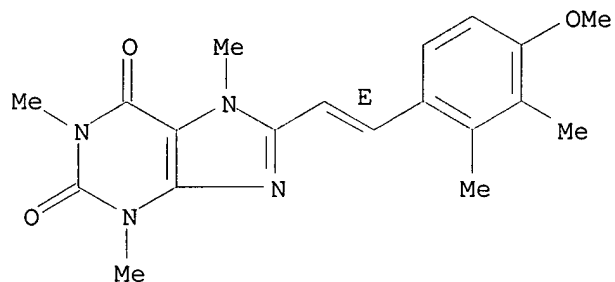
Double bond geometry as shown.



RN 155271-17-3 CAPLUS

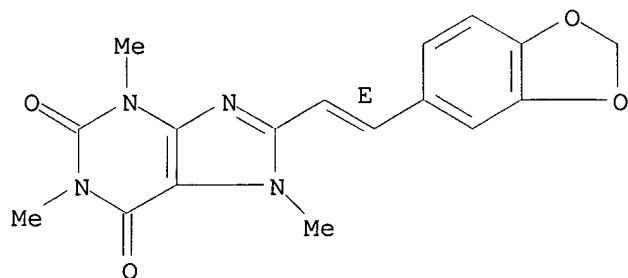
CN 1H-Purine-2,6-dione, 3,7-dihydro-8-[2-(4-methoxy-2,3-dimethylphenyl)ethenyl]-1,3,7-trimethyl-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



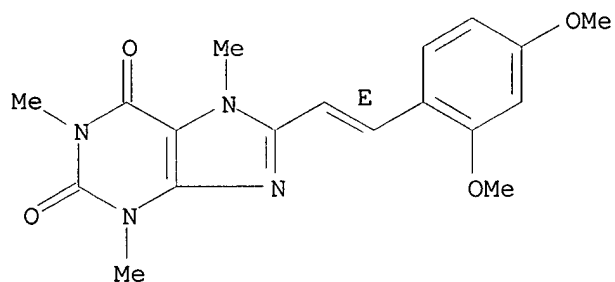
RN 155271-19-5 CAPLUS
CN 1H-Purine-2,6-dione,
8-[2-(1,3-benzodioxol-5-yl)ethenyl]-3,7-dihydro-1,3,7-
trimethyl-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 155271-21-9 CAPLUS
CN 1H-Purine-2,6-dione,
8-[2-(2,4-dimethoxyphenyl)ethenyl]-3,7-dihydro-1,3,7-
trimethyl-, (E)- (9CI) (CA INDEX NAME)

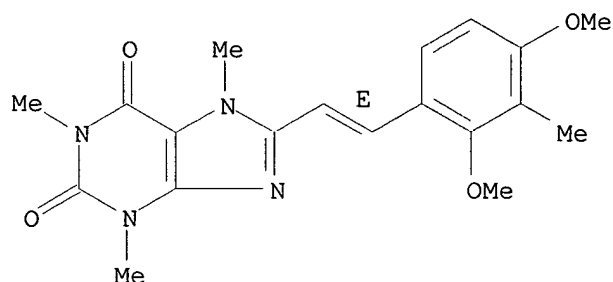
Double bond geometry as shown.



RN 155271-25-3 CAPLUS
CN 1H-Purine-2,6-dione, 8-[2-(2,4-dimethoxy-3-methylphenyl)ethenyl]-3,7-

dihydro-1,3,7-trimethyl-, (E)- (9CI) (CA INDEX NAME)

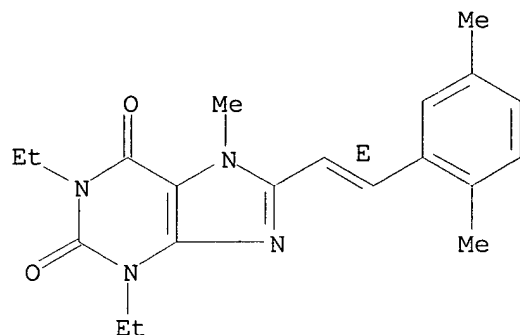
Double bond geometry as shown.



RN 155271-27-5 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(2,5-dimethylphenyl)ethenyl]-1,3-diethyl-3,7-dihydro-7-methyl-, (E)- (9CI) (CA INDEX NAME)

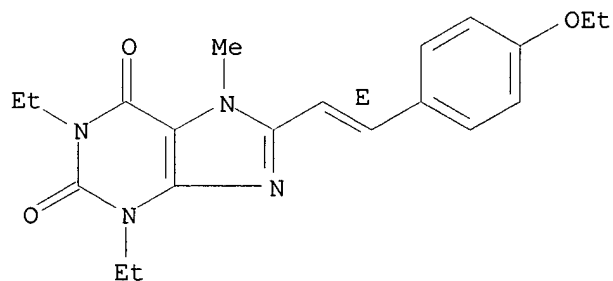
Double bond geometry as shown.



RN 155271-29-7 CAPLUS

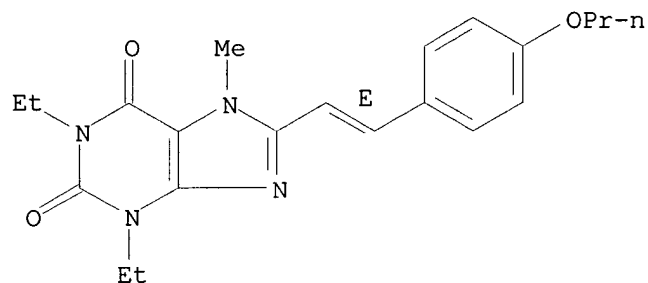
CN 1H-Purine-2,6-dione, 8-[2-(4-ethoxyphenyl)ethenyl]-1,3-diethyl-3,7-dihydro-7-methyl-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



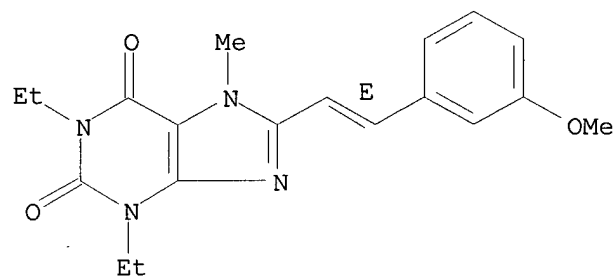
RN 155271-31-1 CAPLUS
CN 1H-Purine-2,6-dione, 1,3-diethyl-3,7-dihydro-7-methyl-8-[2-(4-propoxyphenyl)ethenyl]-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



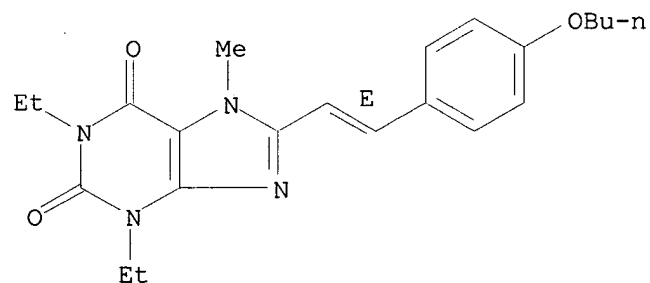
RN 155271-33-3 CAPLUS
CN 1H-Purine-2,6-dione, 1,3-diethyl-3,7-dihydro-8-[2-(3-methoxyphenyl)ethenyl]-7-methyl-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



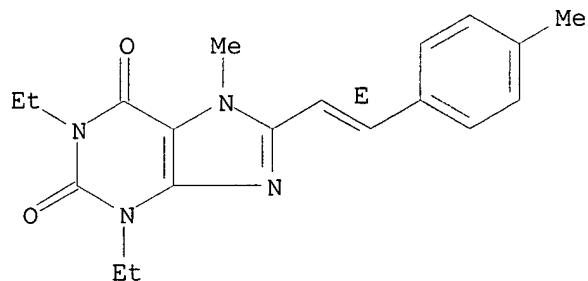
RN 155271-35-5 CAPLUS
CN 1H-Purine-2,6-dione,
8-[2-(4-butoxyphenyl)ethenyl]-1,3-diethyl-3,7-dihydro-
7-methyl-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



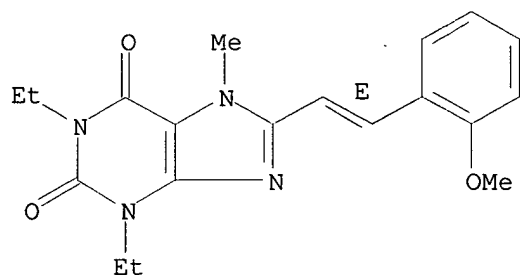
RN 155271-37-7 CAPLUS
CN 1H-Purine-2,6-dione, 1,3-diethyl-3,7-dihydro-7-methyl-8-[2-(4-methylphenyl)ethenyl]-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



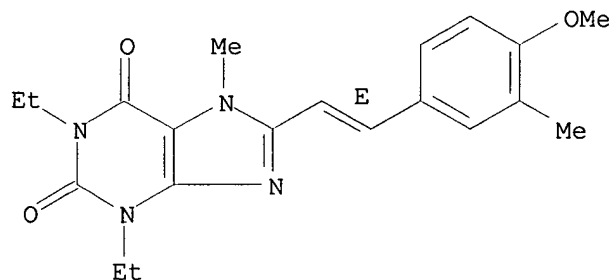
RN 155271-39-9 CAPLUS
CN 1H-Purine-2,6-dione, 1,3-diethyl-3,7-dihydro-8-[2-(2-methoxyphenyl)ethenyl]-7-methyl-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 155271-41-3 CAPLUS
CN 1H-Purine-2,6-dione, 1,3-diethyl-3,7-dihydro-8-[2-(4-methoxy-3-methylphenyl)ethenyl]-7-methyl-, (E)- (9CI) (CA INDEX NAME)

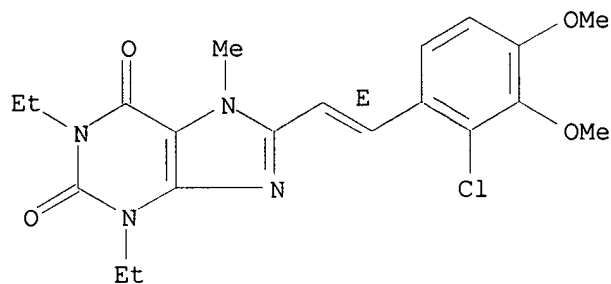
Double bond geometry as shown.



RN 155271-43-5 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(2-chloro-3,4-dimethoxyphenyl)ethenyl]-1,3-diethyl-3,7-dihydro-7-methyl-, (E)- (9CI) (CA INDEX NAME)

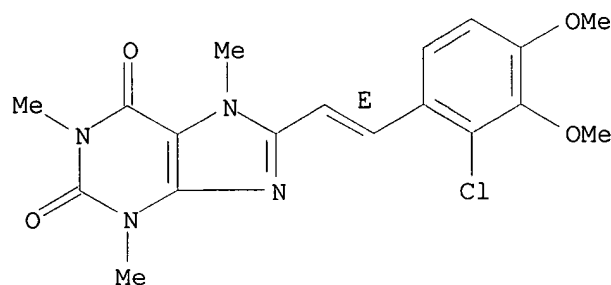
Double bond geometry as shown.



RN 155271-45-7 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(2-chloro-3,4-dimethoxyphenyl)ethenyl]-3,7-dihydro-1,3,7-trimethyl-, (E)- (9CI) (CA INDEX NAME)

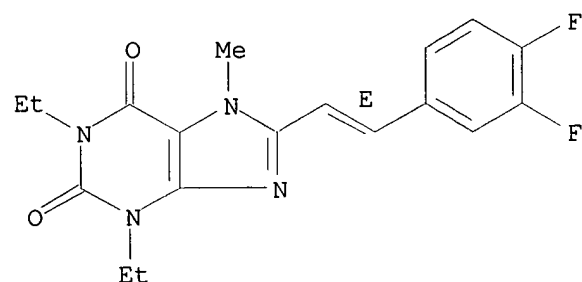
Double bond geometry as shown.



RN 155271-47-9 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(3,4-difluorophenyl)ethenyl]-1,3-diethyl-3,7-dihydro-7-methyl-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

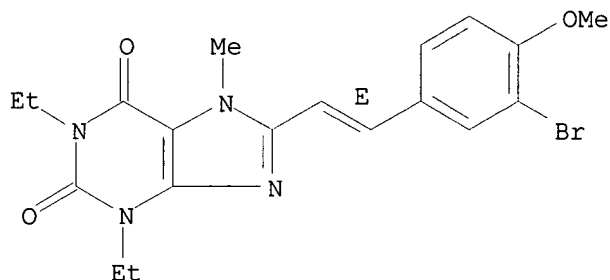


RN 155271-49-1 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(3-bromo-4-methoxyphenyl)ethenyl]-1,3-diethyl-

3,7-dihydro-7-methyl-, (E)- (9CI) (CA INDEX NAME)

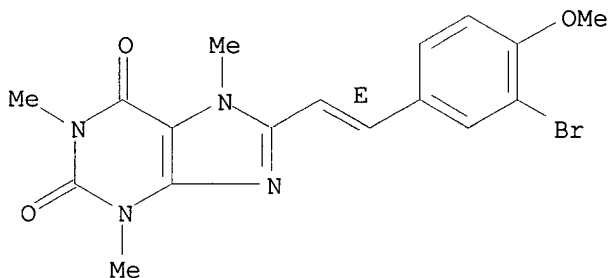
Double bond geometry as shown.



RN 155271-51-5 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(3-bromo-4-methoxyphenyl)ethenyl]-3,7-dihydro-1,3,7-trimethyl-, (E)- (9CI) (CA INDEX NAME)

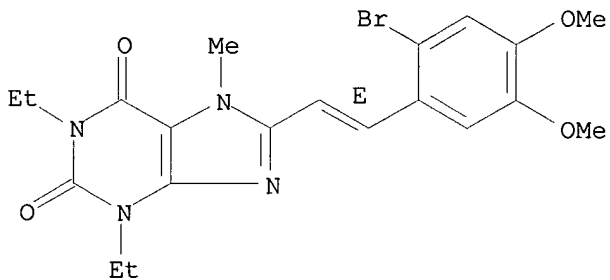
Double bond geometry as shown.



RN 155271-53-7 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(2-bromo-4,5-dimethoxyphenyl)ethenyl]-1,3-diethyl-3,7-dihydro-7-methyl-, (E)- (9CI) (CA INDEX NAME)

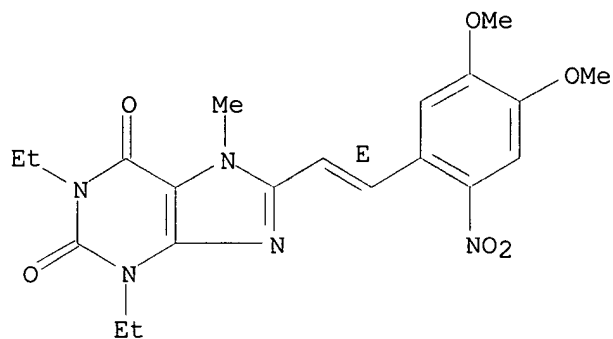
Double bond geometry as shown.



RN 155271-55-9 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(4,5-dimethoxy-2-nitrophenyl)ethenyl]-1,3-diethyl-3,7-dihydro-7-methyl-, (E)- (9CI) (CA INDEX NAME)

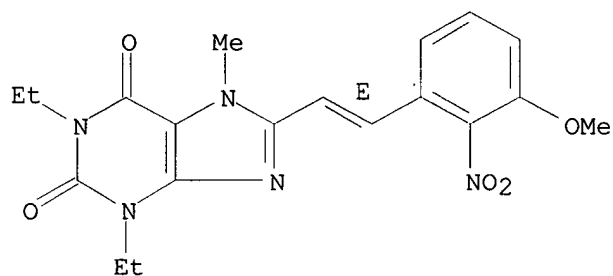
Double bond geometry as shown.



RN 155271-57-1 CAPLUS

CN 1H-Purine-2,6-dione, 1,3-diethyl-3,7-dihydro-8-[2-(3-methoxy-2-nitrophenyl)ethenyl]-7-methyl-, (E)- (9CI) (CA INDEX NAME)

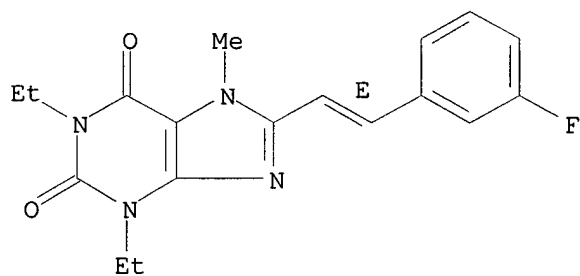
Double bond geometry as shown.



RN 155271-59-3 CAPLUS

CN 1H-Purine-2,6-dione,
1,3-diethyl-8-[2-(3-fluorophenyl)ethenyl]-3,7-dihydro-
7-methyl-, (E)- (9CI) (CA INDEX NAME)

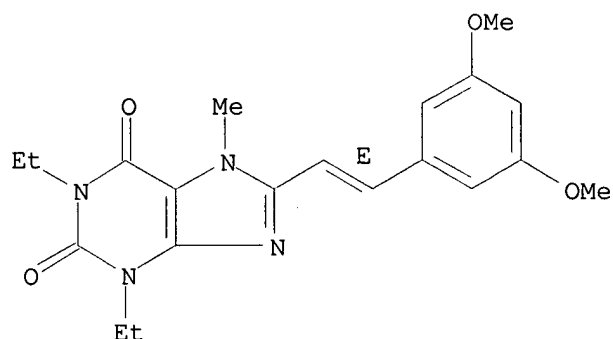
Double bond geometry as shown.



RN 155271-61-7 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(3,5-dimethoxyphenyl)ethenyl]-1,3-diethyl-3,7-dihydro-7-methyl-, (E)- (9CI) (CA INDEX NAME)

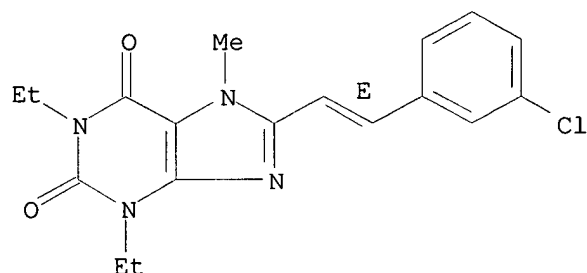
Double bond geometry as shown.



RN 155271-63-9 CAPLUS

CN 1H-Purine-2,6-dione,
8-[2-(3-chlorophenyl)ethenyl]-1,3-diethyl-3,7-dihydro-
7-methyl-, (E)- (9CI) (CA INDEX NAME)

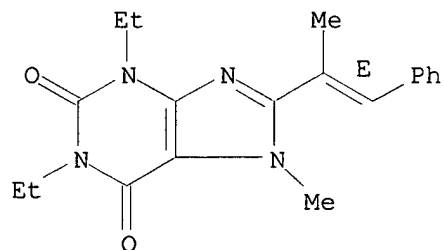
Double bond geometry as shown.



RN 155271-65-1 CAPLUS

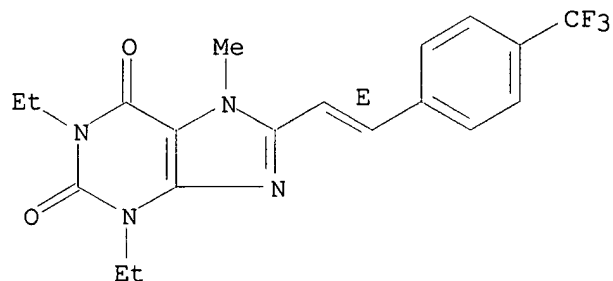
CN 1H-Purine-2,6-dione, 1,3-diethyl-3,7-dihydro-7-methyl-8-(1-methyl-2-phenylethenyl)-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



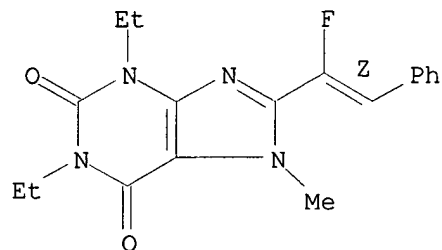
RN 155271-67-3 CAPLUS
CN 1H-Purine-2,6-dione, 1,3-diethyl-3,7-dihydro-7-methyl-8-[2-[4-(trifluoromethyl)phenyl]ethenyl]-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



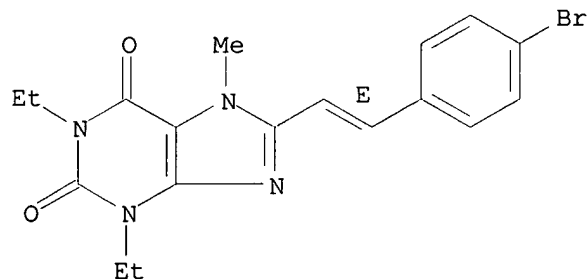
RN 155271-69-5 CAPLUS
CN 1H-Purine-2,6-dione,
1,3-diethyl-8-(1-fluoro-2-phenylethenyl)-3,7-dihydro-
7-methyl-, (Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



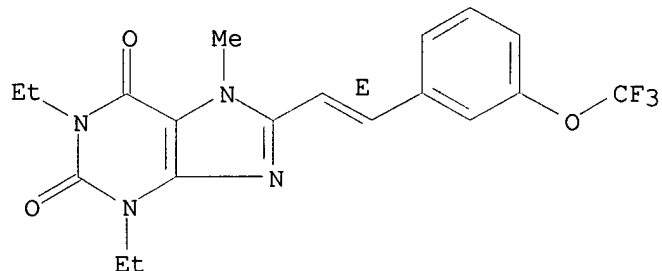
RN 155271-71-9 CAPLUS
CN 1H-Purine-2,6-dione,
8-[2-(4-bromophenyl)ethenyl]-1,3-diethyl-3,7-dihydro-
7-methyl-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



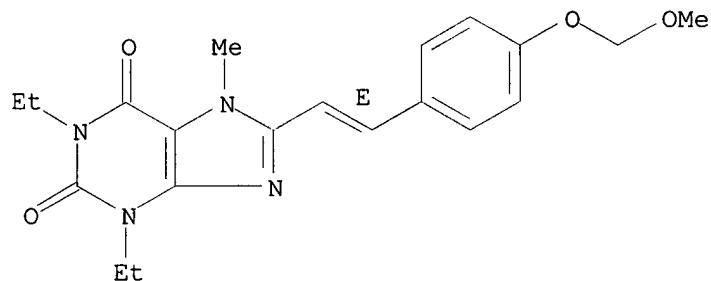
RN 155271-73-1 CAPLUS
CN 1H-Purine-2,6-dione, 1,3-diethyl-3,7-dihydro-7-methyl-8-[2-[3-(trifluoromethoxy)phenyl]ethenyl]-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



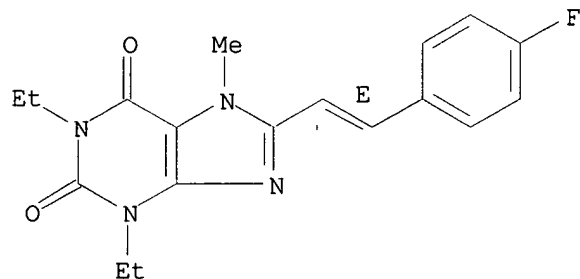
RN 155271-75-3 CAPLUS
CN 1H-Purine-2,6-dione, 1,3-diethyl-3,7-dihydro-8-[2-[4-(methoxymethoxy)phenyl]ethenyl]-7-methyl-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



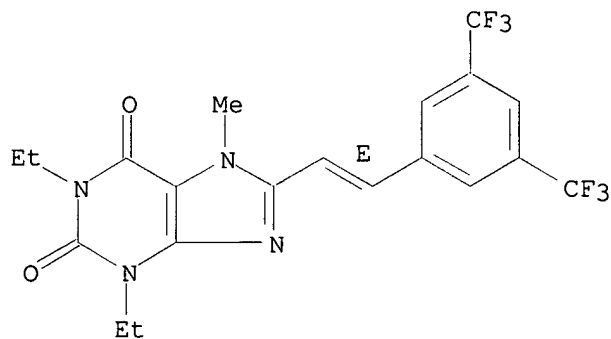
RN 155271-77-5 CAPLUS
CN 1H-Purine-2,6-dione, 1,3-diethyl-8-[2-(4-fluorophenyl)ethenyl]-3,7-dihydro-7-methyl-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



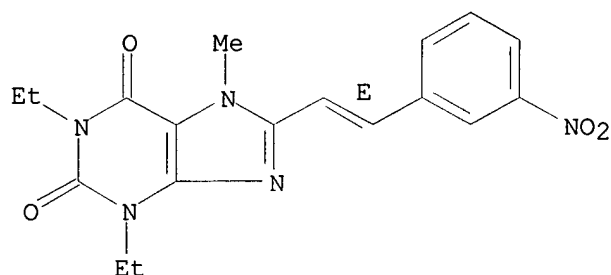
RN 155271-79-7 CAPLUS
CN 1H-Purine-2,6-dione, 8-[2-[3,5-bis(trifluoromethyl)phenyl]ethenyl]-1,3-diethyl-3,7-dihydro-7-methyl-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



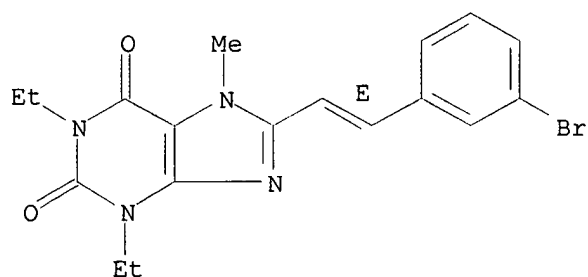
RN 155271-83-3 CAPLUS
CN 1H-Purine-2,6-dione, 1,3-diethyl-3,7-dihydro-7-methyl-8-[2-(3-nitrophenyl)ethenyl]-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 155271-85-5 CAPLUS
CN 1H-Purine-2,6-dione, 8-[2-(3-bromophenyl)ethenyl]-1,3-diethyl-3,7-dihydro-7-methyl-, (E)- (9CI) (CA INDEX NAME)

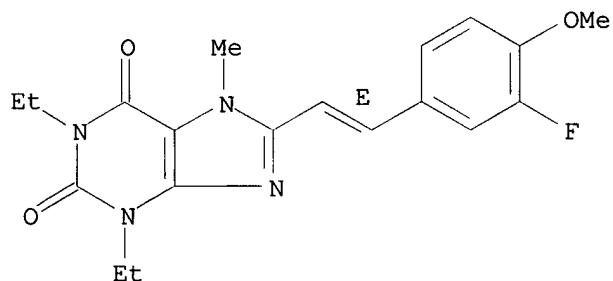
Double bond geometry as shown.



RN 155271-96-8 CAPLUS

CN 1H-Purine-2,6-dione, 1,3-diethyl-8-[2-(3-fluoro-4-methoxyphenyl)ethenyl]-3,7-dihydro-7-methyl-, (E)- (9CI) (CA INDEX NAME)

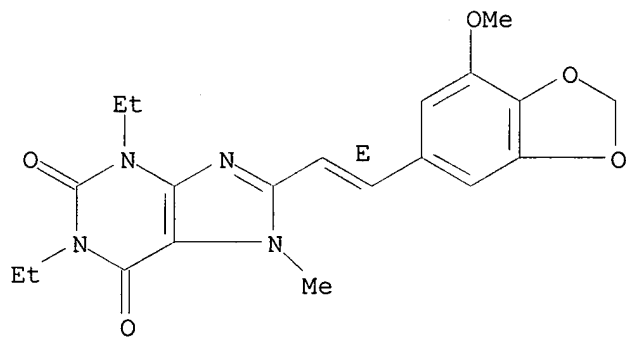
Double bond geometry as shown.



RN 155272-00-7 CAPLUS

CN 1H-Purine-2,6-dione, 1,3-diethyl-3,7-dihydro-8-[(1E)-2-(7-methoxy-1,3-benzodioxol-5-yl)ethenyl]-7-methyl- (9CI) (CA INDEX NAME)

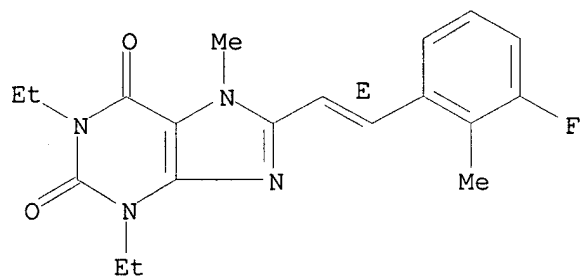
Double bond geometry as shown.



RN 155272-02-9 CAPLUS

CN 1H-Purine-2,6-dione, 1,3-diethyl-8-[2-(3-fluoro-2-methylphenyl)ethenyl]-3,7-dihydro-7-methyl-, (E)- (9CI) (CA INDEX NAME)

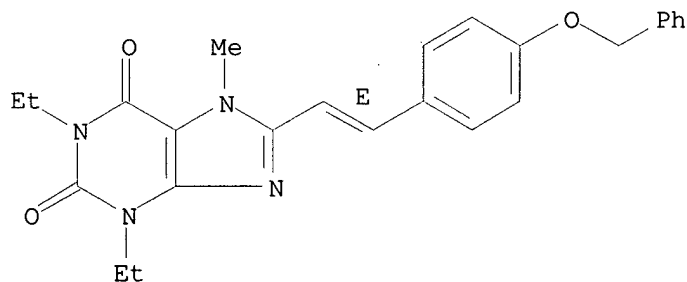
Double bond geometry as shown.



RN 155272-06-3 CAPLUS

CN 1H-Purine-2,6-dione, 1,3-diethyl-3,7-dihydro-7-methyl-8-[2-[4-(phenylmethoxy)phenyl]ethenyl]-, (E)- (9CI) (CA INDEX NAME)

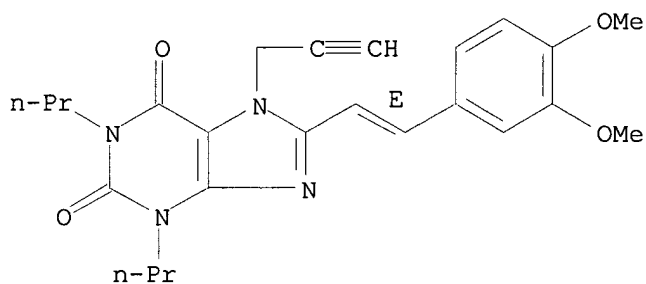
Double bond geometry as shown.



RN 155814-25-8 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(3,4-dimethoxyphenyl)ethenyl]-3,7-dihydro-1,3-dipropyl-7-(2-propynyl)-, (E)- (9CI) (CA INDEX NAME)

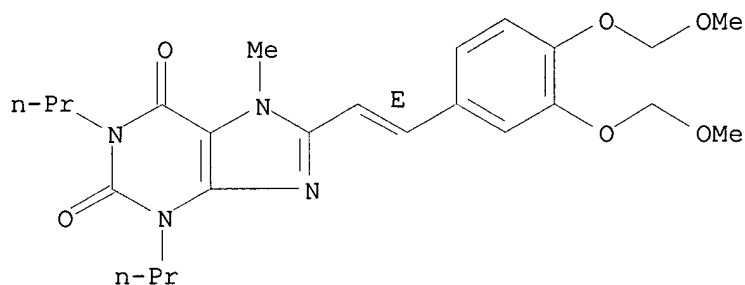
Double bond geometry as shown.



RN 155814-26-9 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-[3,4-bis(methoxymethoxy)phenyl]ethenyl]-3,7-dihydro-7-methyl-1,3-dipropyl-, (E)- (9CI) (CA INDEX NAME)

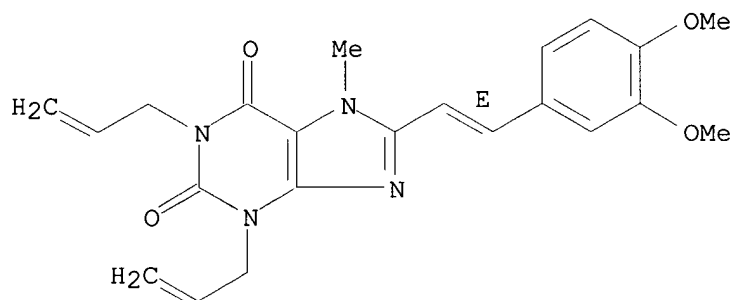
Double bond geometry as shown.



RN 155814-28-1 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(3,4-dimethoxyphenyl)ethenyl]-3,7-dihydro-7-methyl-1,3-di-2-propenyl-, (E)- (9CI) (CA INDEX NAME)

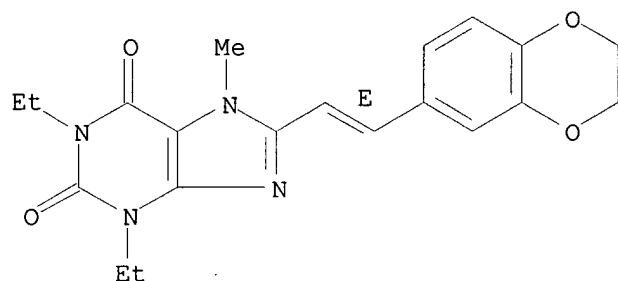
Double bond geometry as shown.



RN 155814-32-7 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(2,3-dihydro-1,4-benzodioxin-6-yl)ethenyl]-1,3-diethyl-3,7-dihydro-7-methyl-, (E)- (9CI) (CA INDEX NAME)

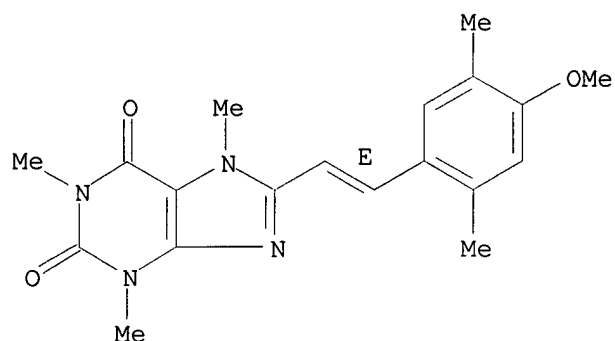
Double bond geometry as shown.



RN 155814-34-9 CAPLUS

CN 1H-Purine-2,6-dione, 3,7-dihydro-8-[2-(4-methoxy-2,5-dimethylphenyl)ethenyl]-1,3,7-trimethyl-, (E)- (9CI) (CA INDEX NAME)

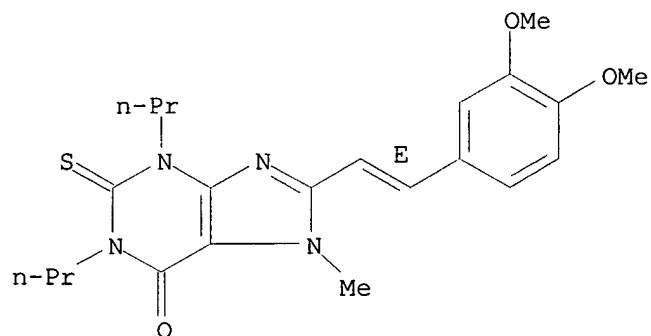
Double bond geometry as shown.



RN 175675-60-2 CAPLUS

CN 6H-Purin-6-one, 8-[2-(3,4-dimethoxyphenyl)ethenyl]-1,2,3,7-tetrahydro-7-methyl-1,3-dipropyl-2-thioxo-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



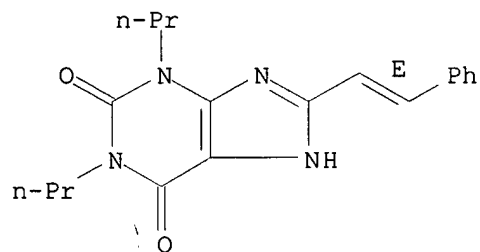
IT 132940-42-2P 141807-95-6P

RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
(prepn. of arylvinylxanthines as selective A2 receptor antagonists)

RN 132940-42-2 CAPLUS

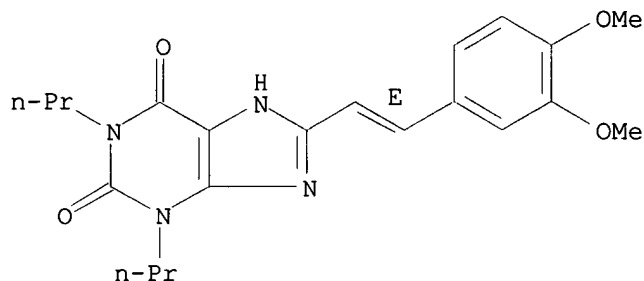
CN 1H-Purine-2,6-dione, 3,7-dihydro-8-[(1E)-2-phenylethenyl]-1,3-dipropyl-, (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 141807-95-6 CAPLUS
 CN 1H-Purine-2,6-dione, 8-[(1E)-2-(3,4-dimethoxyphenyl)ethenyl]-3,7-dihydro-1,3-dipropyl- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

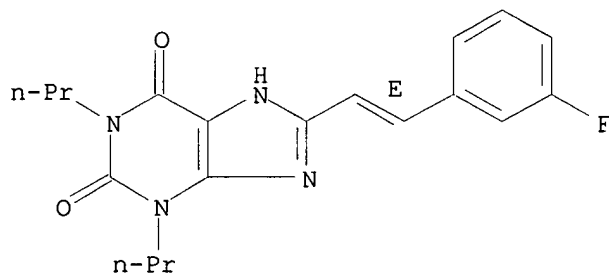


IT 147700-43-4P 147700-45-6P 147700-47-8P
 147700-53-6P 151539-17-2P 151539-20-7P
 151539-24-1P 151539-29-6P 151539-32-1P
 151539-33-2P 151539-35-4P 151539-37-6P
 151539-41-2P 151539-43-4P 151539-48-9P
 151539-51-4P 151539-63-8P 151539-68-3P
 155271-00-4P 155271-08-2P 155271-82-2P
 155271-84-4P 155271-86-6P 155271-88-8P
 155271-90-2P 155271-93-5P 155271-95-7P
 155271-97-9P 155271-99-1P 155272-01-8P
 155272-07-4P 155272-08-5P 155272-10-9P
 155814-27-0P 155814-29-2P 155814-35-0P
 169958-97-8P

RL: RCT (Reactant); SPN (Synthetic preparation); THU (Therapeutic use);
 BIOL (Biological study); PREP (Preparation); USES (Uses)
 (prepn. of arylvinylxanthines as selective A2 receptor antagonists)

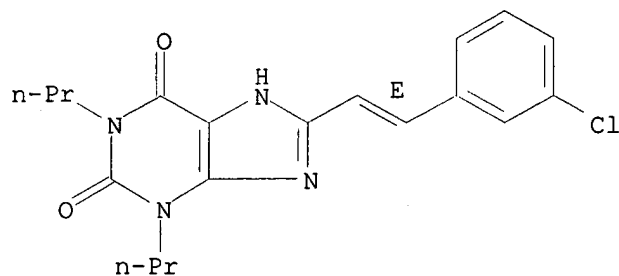
RN 147700-43-4 CAPLUS
 CN 1H-Purine-2,6-dione, 8-[2-(3-fluorophenyl)ethenyl]-3,7-dihydro-1,3-dipropyl-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 147700-45-6 CAPLUS
 CN 1H-Purine-2,6-dione, 8-[2-(3-chlorophenyl)ethenyl]-3,7-dihydro-1,3-dipropyl-, (E)- (9CI) (CA INDEX NAME)

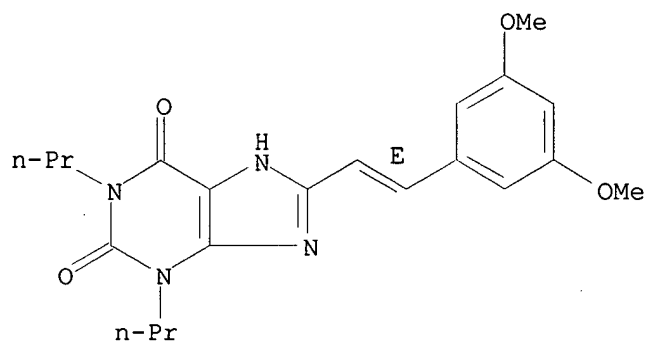
Double bond geometry as shown.



RN 147700-47-8 CAPLUS

CN 1H-Purine-2,6-dione, 8-[(1E)-2-(3,5-dimethoxyphenyl)ethenyl]-3,7-dihydro-1,3-dipropyl- (9CI) (CA INDEX NAME)

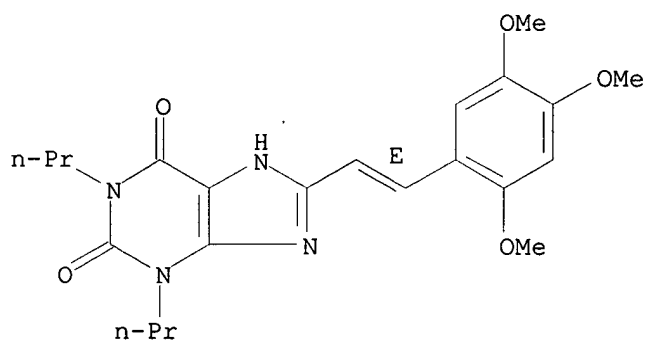
Double bond geometry as shown.



RN 147700-53-6 CAPLUS

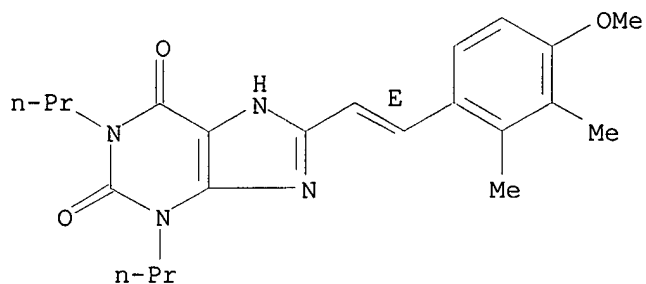
CN 1H-Purine-2,6-dione, 3,7-dihydro-1,3-dipropyl-8-[2-(2,4,5-trimethoxyphenyl)ethenyl]-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



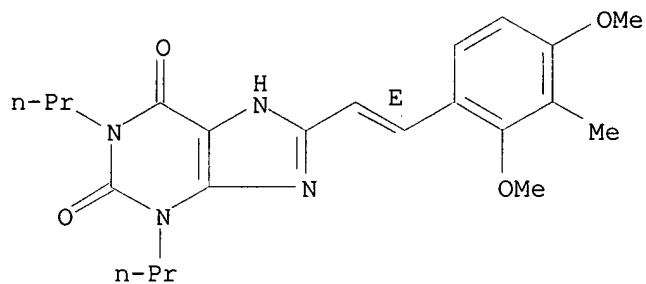
RN 151539-17-2 CAPLUS
CN 1H-Purine-2,6-dione, 3,7-dihydro-8-[2-(4-methoxy-2,3-dimethylphenyl)ethenyl]-1,3-dipropyl-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



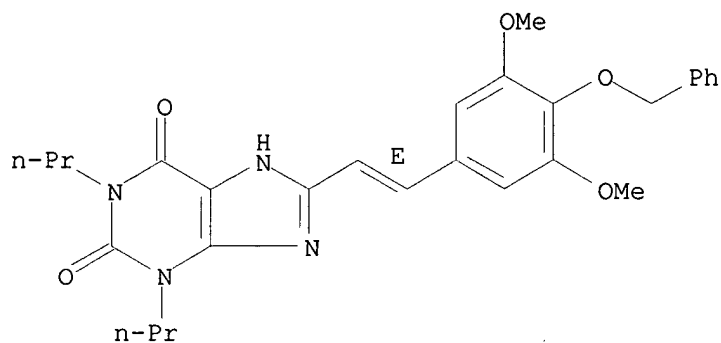
RN 151539-20-7 CAPLUS
CN 1H-Purine-2,6-dione, 8-[2-(2,4-dimethoxy-3-methylphenyl)ethenyl]-3,7-dihydro-1,3-dipropyl-, (E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 151539-24-1 CAPLUS
CN 1H-Purine-2,6-dione, 8-[2-[3,5-dimethoxy-4-(phenylmethoxy)phenyl]ethenyl]-3,7-dihydro-1,3-dipropyl-, (E)- (9CI) (CA INDEX NAME)

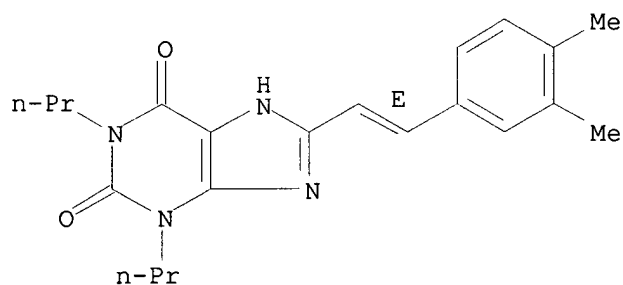
Double bond geometry as shown.



RN 151539-29-6 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(3,4-dimethylphenyl)ethenyl]-3,7-dihydro-1,3-dipropyl-, (E)- (9CI) (CA INDEX NAME)

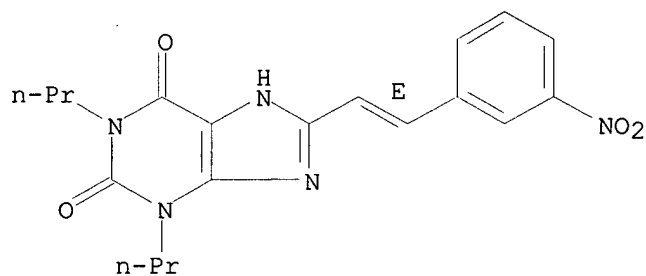
Double bond geometry as shown.



RN 151539-32-1 CAPLUS

CN 1H-Purine-2,6-dione, 3,7-dihydro-8-[(1E)-2-(3-nitrophenyl)ethenyl]-1,3-dipropyl- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 151539-33-2 CAPLUS

CN 1H-Purine-2,6-dione, 8-[2-(2-chlorophenyl)ethenyl]-3,7-dihydro-1,3-dipropyl-, (E)- (9CI) (CA INDEX NAME)